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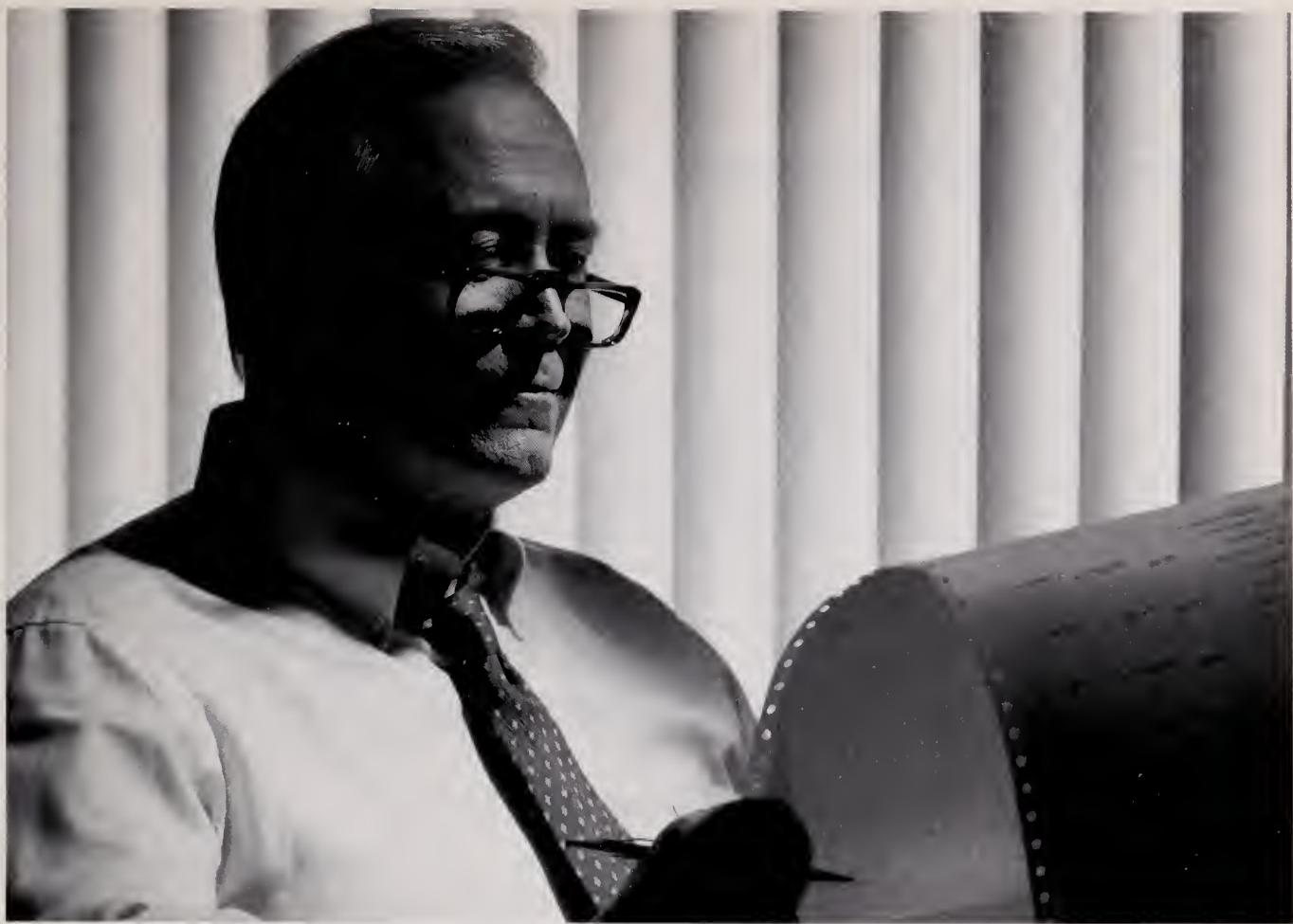
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Cover photograph by Taylor Dabney

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LETTERS

Health forum for teen-agers coming in March

The Medical Society of Virginia and Auxiliary are joining together to sponsor a program titled "Our Youth, Our Future" in Richmond on Tuesday, March 20. The Richmond Centre at the 6th Street Marketplace downtown will be the setting for the all-day program, which will be an Adolescent Health Forum for Virginia's teen-agers; 1,500 to 2,000 ninth to twelfth grade students at schools over the state are to be invited to attend. Lunch and snacks will be provided. Mrs. Eileen Thomas, Portsmouth, is serving with me as cochairman.

The faculty will be composed of physicians, psychologists, well-known sports figures, and other role models and inspirational speakers. Their overall theme will be one of building self-esteem, encouraging the students to live healthy lifestyles and building decision-making skills.

Teenage morbidity and mortality rates have in-

creased 11% in the last 20 years, according to an AMA white paper. There seem to be five contributing categories: sexuality, alcohol/drug abuse, emotional disorders, abuse/victimization, and violence/crime. Poor self-esteem is the common thread running through all these areas. The program will serve to focus on coping with the complexities of maturing but will also affirm the many positive choices these young people are making.

Vivian Petty (Mrs. Carroll T.)

President,
Medical Society of Virginia Auxiliary
6 West Glenbrooke Circle, Richmond VA 23229

A severe critic reveals his identity

A recent editorial refers to a reader who, in his response to the recent Physician Opinion Survey, described the journal as "poor in literary or scientific merit."^{1,2} That reader is I.

You asked, "Where were you when we needed you?" I am going to tell you. I was licking my wounds over the manuscripts rejected by your editorial staff. The last rejection was one entitled, "Robert Burns," a light, fanciful, obstetrical history, which would have been of considerable interest to obstetricians, and

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even more interesting to those lighthearted ones who occasionally like an injection of humor into their scientific journals. Your editorial staff found it had no wit, no humor, and they could not fathom its value to a journal which proposes to be scientific.

During the long days of Pierce Rucker and Harry Warthan, I never suffered a rejection in over 15 contributions I made to the journal.

In conclusion, I would respectfully suggest that your editorial staff is somewhat lacking in humor, the publication lacks eclat. It is dull, flat, uninspired, and indicates an almost complete unawareness to classical references. Worst of all, it has no wit.

I write in the best of good spirit and good wishes for VIRGINIA MEDICAL.

William M. Bickers, MD

12401 Gayton Road, #128
Richmond VA 23233

1. Comments from the Physicians Opinion Survey. Va Med 1989;116:407
2. Kendig ELjr. Commentary. Va Med 1989;116:441

The Editor replies: At present, each article submitted to this journal is reviewed by at least two physicians, one of whom is a member of the same medical specialty as the author. If any reviewer shows any inclination toward publication, we try to suggest ways by which this can be accomplished. As I recall, the article, "Robert Burns," was indeed light and fanciful; the reviewers felt it worthy of publication but unfortunately not suitable for a medical journal.

When Dr. Bickers characterized the journal as without wit, dull, flat, and uninspired, I am sure he was not referring to the February 1988 issue, in which his text on liability litigation was the lead article¹ and was the subject of a special cover, printed in 4-color.

We would like to publish Dr. Bickers' work; most of the time, we will.

1. Bickers WM. The joys of liability litigation. Va Med 1988;115:68-70.

Article on contract problems with HMO strikes a chord

I read with interest the article by Dr. Clifton L. Peay in the November issue of VIRGINIA MEDICAL¹ in regards to his contractual problems with an HMO. It is interesting to see that the contagious HMO disease, which began in Minnesota in the early Seventies and which stimulated my migration to the Northern Neck of Virginia to practice, is infiltrating the metropolitan areas of the State of Virginia. It is my point of view from reviewing with colleagues in Minnesota that out of the fifteen HMOs in that state, only one has been economically viable. It is to the point where the largest HMO in Minnesota reimburses physicians for the care of their patients at less percentage than Medicaid. Again, this represents big business intervention into

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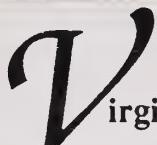
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health care delivery, where the bottom line is the bottom line and not patient care. Once again, the business organizations of the HMOs view physicians primarily as employees to do with as they please in order to maintain profit margins.

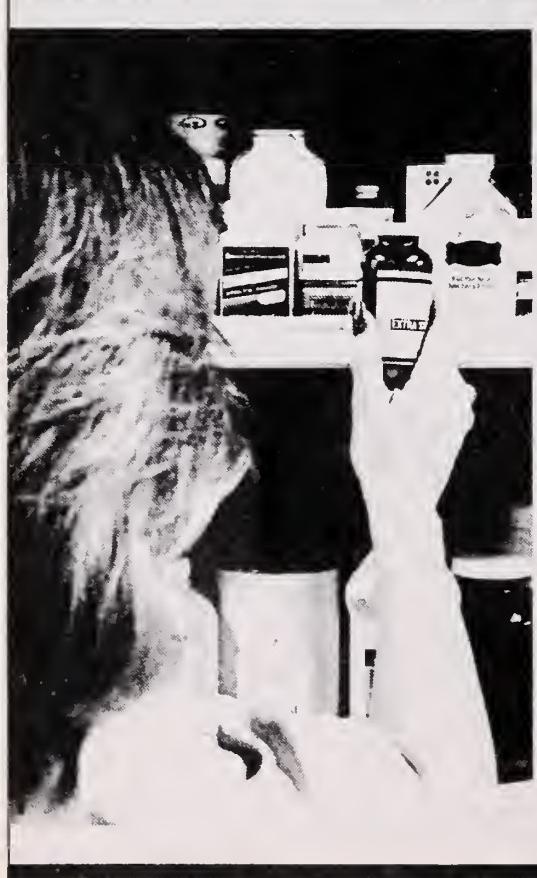
It is hoped that physicians who work in areas of HMOs take the advice of Dr. Peay. Indeed, the contracts should be carefully reviewed, but more importantly, the principle of medical care being delivered by actuarial decree or HMO decree and not by patient need, must be refuted. Nowhere in the Hippocratic oath is business mentioned, and we physicians need to be prudent in this area as well. Now, if we can only do something about the Medicare bureaucracy and the disparity in rural health care in regard to reimbursement by all third-party "non-payers," health care may be back on its feet taking care of the patients instead of big business.

One final note: Ultimately, when all is said and done and the century moves on, those who run big business will throw up their hands and say, "Medicine is not for us," and the only ones left will be the patients and the physicians—the way it was in the beginning.

Timothy A. Mjos, MD

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1. Peay CL. Point of View: Hoist by an HMO's fine print. Va Med 1989;116:466



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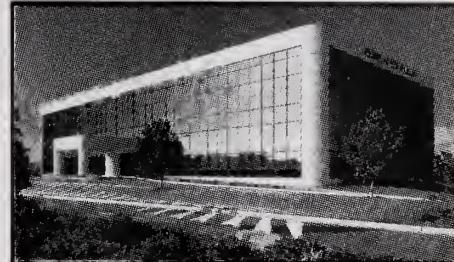
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142nd Annual Meeting

Summarized in these pages are actions taken by the House of Delegates during its second session on November 11 during The Medical Society of Virginia's annual meeting in Richmond. Dr. J. Thomas Hulvey was the presiding officer. Conducting the deliberations of the House were Dr. George E. Broman, Speaker, and Dr. Lawrence K. Monahan, Vice Speaker.

REFERENCE COMMITTEE ONE: Education and Interprofessional Relations

J. Latane Ware, MD, Chairman
Louis D. Parham, Jr., MD, James P. Baker, MD,
Henry D. Patterson, MD, Jack C. Turner, MD,
John T. Hearn, MD, Daniel N. Mohler, Jr., MD,
Joseph V. Puglise, MD, Thomas M. Fulcher, MD,
and Samir Keblawi (Student)

ADDRESS OF THE PRESIDENT

Reference Committee One commended Dr. J. Thomas Hulvey for his "perceptive and outstanding" presidential address, Chairman Ware told the delegates, and recommended that it be distributed to the entire membership. (See page 32.)

The Committee also recommended that Dr. Hulvey be elected an honorary active member of the Medical Society of Virginia, and the House did so forthwith.

ELECTRONIC BILLING

Agreeing with the Reference Committee's endorsement, the House adopted the following six recommendations from the Report of the Electronic Billing Committee:

- 1) That the Society not attempt to initiate an electronic billing service but encourage vendors to enter the market;
- 2) That dialogue with Blue Cross/Blue Shield regarding the electronic

billing issue be encouraged;

3) That a system be developed to keep track of the time required to process paper claims;

4) That the Society urge Blue Cross/Blue Shield to use the information it receives from Medicare on the explanation of benefits to pay claims;

5) That the Society alert hospitals to the fact that information from Blue Cross/Blue Shield data may be sold to them and that hospital staffs should make every attempt to ensure that they are notified when such a transaction takes place;

6) That the Society continue to monitor the activities of Blue Cross/Blue Shield with respect to electronic billing.

ACCESS TO CARE

Four resolutions from the Virginia Academy of Family Physicians had been referred to Reference Committee One for study.

The first called for the Medical Society of Virginia to seek a pilot

Taylor Dabney photographed the annual meeting.

Executive Editor Ann Gray wrote the report of the delegates' actions.



Shown above immediately after their election during the House of Delegates' second session are the Medical Society of Virginia's officers for 1989-1990. Seated in front at left is the President, Dr. William H. Barney, Lynchburg. Seated at right is the President Elect, Dr. John A. Owen, Jr., Charlottesville. Behind Dr. Owen are Dr. Leon I. Block, Falls Church, First Vice President (left), and Dr. William W. Regan, Richmond, Second Vice President. Behind Dr. Barney are Dr. George E. Broman, Culpeper, Speaker of the House (left), and Dr. Lawrence K. Monahan, Roanoke, Vice Speaker, both of whom were reelected. The newly elected Third Vice President, Dr. William H. Sipe, Newport News, was not present for the photograph.

The delegates also elected two new councilors, Dr. Louis D. Parham, Hampton (1st District), and Dr. James A. Shield, Jr., Richmond (3rd District), and two new vice councilors, Dr. Norman R. Edwards, Newport News (1st), and Dr. Lawrence E. Blanchard, Richmond (3rd). The other councilors and vice councilors were reelected.

For a complete listing of the new officers, see page 28.

On the floor of the House: At left, Dr. William J. McAveney, Springfield. Below, from left, Dr. Richard N. Baylor, Kilmarnock, Dr. J. Colin Forrester, Callao, and Dr. Timothy J. Mjos, Kilmarnock.



program to offset the increased liability premiums levied on family physicians and obstetricians who provide obstetrical services to women in underserved areas. The second asked the Society to pursue implementation of a comprehensive plan to ensure health care access for the uninsured.

The Committee heard testimony, Dr. Ware related, that several initiatives regarding access-to-care issues were being conducted by both the Society and the State of Virginia in preparation for the 1990 session of the General Assembly. Accordingly, the Committee believed both of these resolutions should be referred to the MSV State Legislative Com-

mittee. The House so referred them.

The third resolution from the Academy asked the Society to refer to one of its committees the task of considering the implications of the universal health care program being promoted by an organization of physicians. Again the Committee recommended referral to the Society's State Legislative Committee and again the House concurred.

MANAGED CARE PLANS

The fourth resolution from the Virginia Academy of Family Physicians related to utilization of services; it brought half a dozen delegates to the microphones for



In the column at right, at top, Dr. Paul Kaufman, Arlington; center, Dr. Douglas H. Chessen, Newport News, with Dr. Harold W. Felton, Deltaville, behind him; and bottom, Dr. Randolph J. Gould, Norfolk.

discussion and wound up the object of a close standing vote. Reference Committee One had added to its language, as shown below, and it was these amendments that were contested:

RESOLVED, that the Medical Society of Virginia support the concept of appropriate utilization, i.e., that any medical professional reimbursement system that rewards underutilization or overutilization with greater profits is contrary to the best interests of patients and detrimental to the professional ethical behavior of physicians.

One delegate disagreed with the insertion of "or overutilization." Another favored both changes, especially the words "appropriate utilization." Other delegates rose to offer shades of these two opinions. The voice vote on the resolution as amended was too close to call. Vice Speaker Monahan ordered a standing vote and the tellers counted. The resolution as amended was adopted 86-67.

ORGAN DONORS

Health care coverage for organ donors was the concern of a resolution from the Virginia Chapter of the American College of Surgeons. It was the decision of the Reference Committee that "the State Legislative Committee is in the best position to investigate possible funding mechanisms for donor trauma patient's medical care," and the Committee recommended that the resolution be so referred. The delegates adopted the Committee's recommendation.

PRIVATE PRACTICE PARAMETERS

The Reference Committee had added two words, "and therapeutic," to a resolution from Dr. C. Barrie Cook to make it more com-

prehensive, Dr. Ware explained. Thus amended, it read as follows:

RESOLVED, that the Medical Society of Virginia seek introduction at the next meeting of the AMA's House of Delegates of a resolution which would require the officers, member groups and staff of the AMA to involve appropriate members of the federation of organized medicine in the process of developing and approving diagnostic and therapeutic practice parameters affecting more than one specialty.

The amended version was adopted without discussion.

CONSENT CALENDAR

Reference Committee One recommended the adoption of these three items and the delegates did so.

1) Report of the Maternal Health Committee, including this recommendation: That all health providers, especially emergency room personnel and practicing physicians, be ever aware of ectopic pregnancy as a cause of death among young women almost every year in Virginia.

2) A resolution from the Virginia Society of Internal Medicine asking the Medical Society of Virginia to "urge the Medicare carrier in Virginia to maintain accurate and up-to-date lists of physicians who participate in the Medicare program for use by Medicare beneficiaries interested in selecting participating physicians for their medical care."

3) The Lynchburg Academy of Medicine's resolution requesting the Medical Society of Virginia to "oppose the practice of 'hotboxing' or any similar process of dangerous rapid weight reduction and to help inform all state, public, and private schools, as well as recreation departments, of the dangers inherent in this practice and of the opposition of the Medical Society of Virginia to such practices."



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REFERENCE COMMITTEE TWO: Legislation and Regulation

Russell D. Evett, MD, Chairman

Robert T. Davis, MD, Lawrence E. Blanchard III, MD,
George J. Carroll, MD, Henry S. Campell, MD,
William E. Painter, MD, Joel R. Poole, MD,
Donald D. Haut, MD, Joseph H. Early, Jr., MD,
Richard E. Linde, MD, and Mike Williams (Student)

TOBACCO USE

Reference Committee Two's report led off with a brace of resolutions relating to tobacco use. Chairman Russell Evett asked the delegates to turn their attention first to a lengthy resolution introduced by the Arlington and Fairfax Counties Medical Societies; it incorporated five Resolved clauses, four of which the Reference Committee recommended adopting forthwith, and the delegates did so, as follows:

- 1) That the Medical Society of Virginia endorse the AMA goal of a smoke-free society by the year 2000;
- 3) That the Medical Society of Vir-

ginia specifically support legislative efforts to restrict or eliminate tobacco smoke in public indoor spaces in order to protect Virginians from the hazards of passive smoke inhalation;

4) That the Medical Society of Virginia oppose any legislation which constrains local governing bodies from adopting more restrictive ordinances relative to smoking to protect their citizens;

5) That the Medical Society of Virginia oppose any legislation designed to force repeal of any ordinances already in place to protect the public from secondhand smoke.

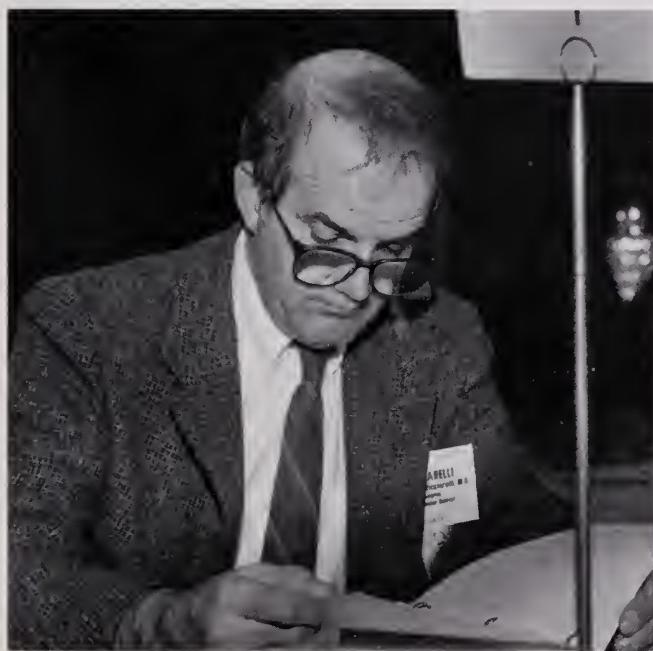
The original Clause 2 had read, "That the Medical Society of Virgin-

ia support and seek passage of legislation in the General Assembly to restrict tobacco use in Virginia." The Reference Committee had developed this substitute clause, Chairman Evett reported, and recommended its adoption: "That the Medical Society of Virginia support public and private programs to discourage the use of tobacco products in Virginia."

At once there were delegates at the mikes, asking for recognition. Support for the extant programs is not what's needed, they said; what's needed is legislation. But there was agreement that the words "and seek" in the original resolution might well be omitted, since the Society should not be asked to introduce legislation. After considerable discussion, the original Clause 2, minus only the words "and seek," was adopted by the delegates as follows:

RESOLVED, that the Medical Society of Virginia support passage of legislation in the General Assembly to restrict tobacco use in Virginia.

Dr. Evett continued with other items on tobacco use. It was the Committee's consensus, he reported, that the Cancer Committee's recommendation to support appropriate



Dr. Giacomo A. Ricciarelli,
Hampton



Dr. James E. Nevin III, Danville,
and Dr. H. S. Campell, Martinsville

clean air legislation in the 1990 General Assembly had been adequately covered by the resolution just adopted, as had a resolution from the Virginia Chapter, American Academy of Pediatrics, and the Virginia Pediatric Society asking the Society to seek a Virginia Clean Indoor Air Act. Accordingly, Reference Committee Two did not recommend action on those measures.

But the Committee had decided that a resolution on tobacco products from the Hampton Medical Society merited adoption, the chairman said, although it needed some amending, as follows:

RESOLVED, that the Medical Society of Virginia seek legislation which would prohibit oppose the sale of tobacco products to children under the age of 18, prevent oppose access by children under the age of 18 to vending machines containing tobacco products, and which would prohibit oppose the use of tobacco products by children under the age of 18 in public places, including schools and school grounds.

Dr. Evett explained that the Committee fully supported prevention of tobacco addiction in children and opposition to the sale of tobacco products to minors, but it was felt that the laws already in place made it difficult to draft additional legislation.

The resolution as amended was adopted.

RURAL HEALTH

The Report of the Rural Health Committee had been referred for consideration to Reference Committee Two. It carried the recommendation that the Medical Society of Virginia adopt 14 statements "as a portion of its position and policy on rural health matters in Virginia and the nation."

The Reference Committee heartily endorsed the efforts of the Rural Health Committee to provide specific methods of addressing the problems of rural physicians, Dr. Evett

MEDICAL SOCIETY OF VIRGINIA
BUDGET 1988-1989

Salaries	\$495,000
Stationery and Supplies	19,000
Office Equipment: Repairs and Replacement	10,000
Building Expense	5,000
Building Maintenance/Repairs/Improvements	45,000
Telephone	14,000
Postage	20,000
Convention Expense	40,000
Council and Committee Expense	20,000
Delegates to American Medical Association	55,000
Staff Travel—AMA	6,000
President's Expense	14,000
Presidential Expense Allowance	15,000
President Elect Expense Allowance	3,000
Staff Travel Expense	8,000
Preparation and Distribution of VIRGINIA MEDICAL	105,000
Editor, VIRGINIA MEDICAL	2,400
Legal Expense	110,000
Legislative Program	35,000
Walter Reed Commission	1,000
MSV Auxiliary	7,000
Membership Dues, Affiliated Agencies	1,000
Special Appropriations	
AMA/ERA	1,000
Scholarships	
Medical College of Virginia	1,000
University of Virginia	1,000
Eastern Virginia Medical School	1,000
Rural Health	1,000
Student Medical Societies	
Medical College of Virginia	4,000
University of Virginia	4,000
Eastern Virginia Medical School	4,000
Virginia Health Council	1,000
Hospital Resident Section	1,500
Other Special Appropriations	10,250
Indigent Care Survey	5,000
Injured Infants Act Study	6,487
Insurance Premiums	17,000
Marketing	5,000
Professional Liability and Tort Reform	80,000
Campaign Expenses	15,000
Newsletter	7,000
Employee Benefits	
Retirement Fund	122,500
Blue Cross/Blue Shield, Life, and Disability	25,000
Payroll Taxes	35,000
Continuing Medical Education	18,000
Consulting Services	
Legislative and Public Relations	65,000
Insurance Actuary	18,000
Physicians' Health and Effectiveness Program	24,776
Part-Time Medical Director	26,250
Miscellaneous	5,000
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TOTAL	\$1,556,163

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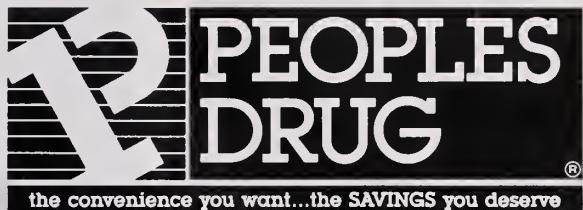
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WINNERS

As the only physician in Victoria, Virginia, Dr. Lewis W. Bridgforth has been on call day and night for 23 years, answering as doctor, adviser, and friend. A thoroughly modest man, he has always clung to the notion that most of what he has done for the people of Victoria was known only to those he has helped. In 1989, that notion went out the window.

First the Victoria Chamber of Commerce surprised him with its Citizen of the Year Award, complete with ceremonial dinner, engraved plaque, prolonged applause, popping flash bulbs—the whole public recognition bit. So acute was Dr. Bridgforth's consternation that he "could hardly make it to his feet," one witness said.

As it turned out, that was a practice run for the Victoria physician's second surprise of the year. This time the setting was the Medical Society of Virginia's annual meeting banquet, with Dr. J. Thomas Hulvey, outgoing MSV President, presiding.

"For all the technological advances that make us better doctors, medicine is still a very human profession," Dr. Hulvey began, "and the spirit of the country doctor lives on. We are more than just mechanics for the body; we are here to comfort, to listen, and to advise. The recipient of this year's A. H. Robins Community Service Award embodies these qualities." Dr. Hulvey launched into some specifics.

The winner's contributions to the children of his community include giving free physicals to members of the local high school's athletic teams, as well as to local Little League and Midget Football teams, "a huge commitment of time," Dr. Hulvey observed, "yet he has been doing it for over 20 years. He holds clinics for children on his free afternoons, and turns his farm over to the



Winner Bridgforth and wife Betty

Boy Scouts for wilderness weekends and jamborees."

He goes far beyond "just treating skinned knees. He provides a positive role model at a time when kids need honest adults to look up to," Dr. Hulvey continued, and he cited this quote from a member of the staff at the local high school: "He has always been available to the needs of our athletes, always willing to come out late at night, when we return from a game, to examine the kids. In the eyes of the youth of the county,

he is the hero of the community."

The award winner is "still on call 24 hours a day," Dr. Hulvey related, "and his extended office hours are for more than just medical help; patients come to him for family, legal and financial advice. Not long ago a family called at 11:30 at night in the midst of a family crisis. The doctor invited them into his home and helped them work through the problem until 2:00 in the morning."

Finally, although the winner has never held local office, "he has been

around whenever the community needed leadership," Dr. Hulvey related. "He was instrumental in the development of a local county bank and has served as an officer and director."

"Now," concluded Dr. Hulvey, "it is my honor to say thank you on behalf of the Medical Society of Virginia and bestow this year's Community Service Award on a man born here in Virginia, educated at Hampden-Sydney, graduated from the Medical College of Virginia, and an outstanding citizen of Victoria, Virginia—Dr. Lewis W. Bridgforth!"

This time Dr. Bridgforth got to his feet without noticeable difficulty, waded swiftly through his applauding peers to the dais, ducked his head in thanks to Dr. Hulvey, and turned to meet the flash bulbs. Clearly, he was getting the hang of it.

A native of Crewe, Virginia, Dr. Bridgforth established his medical practice in Victoria, a rural community (pop. 2,004) much like the one in which he grew up, in 1966. He was president in 1985 of the Southside Medical Society and is the 26th recipient of the Community Service Award.

Awarded the Challenge Cup at the annual MSV golf tourney on the Country Club of Virginia's course was **Dr. Gerald C. Burnett**, South Boston, who carded the low net of 72. One shot off the pace for second place was **Dr. Richard N. Baylor**, Kilmarnock, with a low net of 73. **Dr. John T. Hearn** of Penn Laird, who posted a low gross of 74, won first prize in that category. Other prizes went to **Dr. C. B. Courtney, Jr.**, Newport News, who had the longest drive on #8, **Dr. Norman R. Edwards**, also Newport News, who was closest to the pin on #7 green, and **Dr. Victor N. Guerrero**, Manas-

sas, who was closest to the pin at the 16th hole.

The **Robert P. Trices** of Richmond swept the tennis tourney, with Dr. Trice winning the men's singles, then joining his wife, Edmonia, to take the mixed doubles title.

The commercial exhibits hall was loaded, with handouts and prizes galore from 73 exhibitors.

At the John P. Pearl & Associates booth, **Dr. Henry A. Yancey**, Richmond, won the men's watch, and **Dr. Clyde H. Dougherty**, Hopewell, won the ladies' watch. The 7½-pound cookbook, *Dining at the Homestead*, raffled off by Saint Albans Psychiatric Hospital was won by **Dr. John A. Owen, Jr.**, Charlottesville, who also won an election at the meeting, as see page 11.

The briefcase offered by Sentient Systems was won by **Dr. James L. Van Dyke**, Galax. At the Miles Pharmaceuticals booth, **Dr. Emmett C. Mathews**, Richmond, won a Sony Walkman; the Old Timey Radio raffled by the Haycraft Insurance Agency was won by **Dr. Gerald Weitzman**, Portsmouth; and at the First Insurance Resources booth, a portable TV was won by **Dr. Ganesh Nirmul**, Suffolk.

The prize at the E. R. Squibb booth was a big basket of Virginia products; it was won by **Dr. Norman R. Tingle, Jr.**, Lively. Offered as prizes at the booth of W. B. Saunders were two medical books; they went to **Dr. Jack C. Turner**, Danville, and **Dr. Albert L. Roper**, Norfolk. Two dozen physicians won bottles of wine at the Home IV Care & Nutritional Services booth.

In turn, the Medical Society of Virginia conducted a drawing for the reps in the exhibit hall. Lisa Moreland of the Upjohn Company held the winning ticket and took home the prize, a Virginia ham.

reported, but it felt that such a broad range of recommendations could not be dealt with effectively in a reference committee setting. Accordingly, its members felt the 14 recommendations should be referred to Council for assignment to appropriate committees.

The House agreed and referred as recommended.

Also referred to Council was a resolution relating to a position paper from the Virginia Academy of Family Physicians and "addressing five major barriers to increasing the number of well-trained primary care physicians in Virginia's rural areas." The Resolved clause asked the Medical Society of Virginia to adopt the position paper and work toward reducing the cited barriers. It was noted by the Reference committee that the Society is in the process of analyzing data from a recent survey of primary care physicians and obstetricians throughout the state regarding access to care.

FIREARM LEGISLATION

From the Richmond Academy of Medicine came this resolution supporting firearm legislation: **RESOLVED**, that the Medical Society of Virginia wishes to be on record as favoring methods which control this outbreak of misuse and violent use of firearms, particularly assault and military weaponry.

The preamble to the resolution cited the easy availability and increasing use in Virginia of lethal weapons and gave the estimated cost of care for gunshot wound victims at \$10,000-\$15,000 per incidence. Dr. Warren W. Koontz, Academy president, spoke in support of the resolution, citing a study of 535 patients with renal trauma admitted to the Medical College of Virginia (Va Med December 1989). The study's data indicated a decrease in the rate of total nephrectomy except in patients with gunshot wounds; in these cases, a persistently high rate prevailed, re-



At Reference Committee Two's hearing, Dr. William A. Hazel, Jr., Herndon, at top, and at center, from left, Dr. George A. Hurt, Lynchburg, Dr. Richardson Grinnan, Richmond, and Dr. Donald E. Sly, Norfolk. In the hallway outside the hearing, bottom, are Dr. Michael W. Russell, Culpeper, and Dr. Harry C. Kuykendall, Alexandria.

flecting, the study suggested, the proliferation of increasingly lethal weaponry.

The Reference Committee was in favor of adopting the resolution, its Chairman reported, but recommended deleting mention of an outbreak. A spirited discussion ensued in the House on the proposed "outbreak" deletion and also on the semantics of "assault and military weaponry," but the delegates wound up by adopting the resolution as written.

A resolution on the protection of children from firearms introduced by the Virginia Chapter, American Academy of Pediatrics, and the Virginia Pediatric Society offered two Resolved clauses. One sought "legislation to restrict the availability of firearms, particularly to minors" and the other asked for legislation making the owner of a firearm liable "if the firearm is accessible to a person under 21 and is used in a crime by or involved in unintentional injury of a minor."

As drafted, the resolution was deemed by the Reference Committee to be overbroad, and adoption was not recommended. The House so voted.



INJURED INFANTS ACT

Introduced by the Virginia Academy of Family Physicians was a resolution asking the delegates to adopt another position paper developed by the Academy regarding the Virginia Injured Infants Act.

"Reference Committee Two supports the position paper developed on the subject by the Medical Society of Virginia's State Legislative and Professional Liability Committees," Dr. Evett told the delegates, "and does not feel it would be appropriate to adopt an alternative paper in its place." The Committee therefore recommends, he continued, that the resolution be referred to the Professional Liability Committee. The Chairman noted that testimony at the Committee's hearing indicated the two organizations' position papers were similar in content. The House voted to refer.

DRUNK DRIVING

A resolution from the Norfolk Academy of Medicine had to do with

Top right, Dr. Charles J. Gueriera, Manassas, in the Richmond Marriott's lower lobby. Conversing in the same corridor are, at center, Dr. Randall H. Suslick, Chase City, and Dr. Ronald K. Davis, Richmond; and at bottom, Dr. Lockhart B. McGuire, Charlottesville, and Dr. Eugene F. Poutasse, Richmond.

revoking the driver's licenses of Virginians who were found to have high blood alcohol levels or who refused blood alcohol tests.

The Reference Committee endorsed the resolution's intent but amended it slightly so that it did not suggest that the Society introduce legislation. As amended by the Committee and adopted by the delegates, it read:

RESOLVED, that the Medical Society of Virginia support legislation which would enact administrative driver's license revocation for drivers whose blood alcohol content exceeds the legal limit or who refuse a blood alcohol determination.

PRIVATE REVIEW

The Norfolk Academy of Medicine had introduced a resolution relating to regulation of private review; it was prompted, stated the preamble, by the increasing frequency with which insurance companies include contractual requirement for precertification and length of inpatient stay. These requirements, the resolution stated, "are becoming more and more complicated, difficult and time-consuming for physicians and their staffs to manage."

The resolution asked for relief through legislation or regulation requiring insurance companies to 1) retain sufficient personnel to perform such review; 2) make this personnel "immediately accessible" to their subscribers' physicians, rather than placing the task of obtaining such approval on the physicians; and 3) provide notarized releases in advance authorizing physicians to disclose information about their subscriber-patients over the telephone, "in order to protect the privacy of the patient and the integrity of the physician."

A worthy concept, Reference Committee Two agreed, but it felt the State Legislative Committee best qualified to determine what type and level of regulation would be appropriate. The House concurred and referred the resolution accordingly.

PEER REVIEW

Easement of peer review requirements relating to emergency surgery was the goal of a resolution from the





The photographs on these pages were taken in the Exhibit Hall. Above, Dr. Frederick W. Parker III, Manassas, with Mike Wysocki of Searle Laboratories. Below, Dr. Clarke Russ, Virginia Beach looks over the titles at the W. B. Saunders booth while rep Ginny Mackie stands by.



Medical Society of Virginia's Council. Reference Committee Two recommended the change of one word before adopting. The amended resolution:

RESOLVED, that the Medical Society of Virginia submit a resolution to the American Medical Association House of Delegates which would request the AMA to encourage the Health Care Financing Association to amend the PRO Third Scope of Work to allow at least two working days to obtain an authorization number for emergency surgery when such surgery requires pre-procedure review.

The House adopted the resolution as amended.

PHYSICIAN REIMBURSEMENT

Once again resolutions from the Virginia Academy of Family Physicians appeared in Reference Committee Two's report. One measure (Resolution O) had to do with inequities in Medicare reimbursement between primary care and procedure specialists, the other (Resolution T) asked for the Medical Society of Virginia to conduct a study of the Harvard Resource-Based Relative Value Scale (RBRVS).

After hearing a lot of testimony about these two resolutions, reported the Chairman, the Committee had decided that neither resolution should be adopted, due to the fact that both subjects are being considered at the state level by the MSV Committee on Federal Legislation and at the national level by the AMA.

The discussion was lively pro and con. Out of it came the adoption of this significantly amended version of Resolution O:

RESOLVED, that the Medical Society of Virginia direct its State and Federal Legislative Committees to support the AMA's position on the Harvard RVRBS.

That cancelled the need for Resolution T and it was not adopted.

OBSTETRICAL CARE

After spirited discussion, Reference Committee Two's recommended amendments to a resolution on obstetrical care were rejected by the delegates and the original reinstated for passage. As framed by the Virginia Academy of Family Physicians and adopted by the House, it read:

RESOLVED, that the Medical Society of Virginia urge the state legislature to provide assistance to physicians presently providing obstetrical care without reimbursement or with limited reimbursement. The Committee's amendments spoke of "incentives," and the House was in no mood for incentives, it wanted assistance for these doctors.

HEALTH INSURANCE

There was quite a little debate, too, on a resolution on child health insurance reform introduced by the Virginia Chapter, American Academy of Pediatrics, and the Virginia Pediatric Society. It proposed mandating a package of preventive health care services for children in every health insurance policy written in Virginia.

Reference Committee Two did not recommend adoption of the measure, Chairman Evett explained, because of testimony that "the existing moratorium on mandated benefits in Virginia in response to the substantial cost increases that have resulted from a steady rise in the number of mandated benefits, and these increased insurance costs have led to a surge in the number of uninsureds." Therefore, although the Reference Committee recognized the desirability of adequate insurance coverage for the care of children, it had concluded that it was not practical to recommend any additional mandated benefits at this time.

After some discussion, the delegates first amended the resolution so that the insurers would be required to offer, rather than include, the desired coverage and then voted to



Above, Jim Thomson of Merck, Sharpe & Dohme listens to Dr. Sigsby W. Gayle, Richmond, Exhibits Chairman.

Below, Dr. Kang H. Rah, Richmond, signs in for a prize at the John Pearl & Associates booth as Gary Pearl and Mike Huennekens look on.





While Medical Society of Virginia members were conducting their annual meeting, the MSV Auxiliary was holding its midyear board meeting, in which the two women above had leading roles. At right is Mrs. Carroll T. (Vivian) Petty, Richmond, MSVA President 1989-90. With her is the President Elect Mrs. Victor N. (Heidi) Guerrero, Manassas.

refer the matter to the State Legislative Committee.

Another resolution from the two groups of pediatricians dealt with "limited access to comprehensive, continuous primary health care and services by virtue of the inadequacy of Medicaid reimbursement for such services" and asked the Medical Society of Virginia to seek from the General Assembly additional budget appropriations that would raise reimbursement to usual, customary and reasonable levels. On the recommendation of the Reference Committee, the House referred this resolution also to the State Legislative Committee.

From the Virginia Chapter of the American College of Surgeons came a resolution asking for a legislated funding mechanism to pay for medical care given uninsured persons injured in motor vehicle accidents.

Again the Reference Committee recommended referral to the Society's State Legislative Committee, but this time sent with it the authority to take action. The House referred as recommended.

DRUG DISPENSING

Next on Reference Committee Two's agenda was a resolution from the Norfolk Academy of Medicine calling for regulatory legislation of bulk sales of scheduled drugs. The Committee favored adoption, the Chairman reported, but recommended changing one word, as follows:

RESOLVED, that the Medical Society of Virginia seek support legislation to authorize the Virginia State Boards of Medicine and Pharmacy to regulate bulk sales of scheduled drugs.

The House adopted the resolution as amended.

DENIAL OF CLAIMS

These two Resolved clauses culminated the Roanoke Academy of Medicine's submitted resolution on denial of claims:

RESOLVED, that the Medical Society of Virginia request the State Commissioner of Insurance to instruct health insurers, when communicating to their clients a lack of coverage, to strenuously avoid the implication that the physician's charges were excessive, but instead plainly and simply state that the policy does not cover all the charges generated.

RESOLVED, that the Medical Society of Virginia recommend to the Commissioner that the health insurers be instructed to strenuously avoid implying that the charges generated in the claim may not have been medically necessary.

It was Reference Committee Two's observation that "it is not practical to look to the Insurance Commissioner to force changes in these behavior patterns," and the Committee recommended that both clauses be amended to delete the Commissioner from the equation and instead "urge the insurers" to refrain from these derogatory comments.

Numerous delegates rose to speak to the measure. All wanted the original proposal, some wanted slight changes here or there. The consensus finally reached was that the original resolution be referred to Council with a request for action.

GENERIC DRUGS

The House also wanted action on a resolution from the Virginia Academy of Family Physicians asking the Medical Society of Virginia to adopt a group of recommendations on generic drugs relating particularly to therapeutic substitution and, on Reference Committee Two's recommendation, referred it for that action to the Society's Therapeutics and Devices Committee.

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ANIMAL RESEARCH

On the recommendation of the Reference Committee, the House adopted without discussion a resolution from the Animal Research Committee asking that the Medical Society of Virginia support legislation making it a federal crime to attempt to damage or destroy any animal research facility.

CONSENT CALENDAR

Reference Committee Two recommended the adoption on a consent calendar basis of the following three items, and the House so adopted.

1) A resolution on health care savings accounts from the Virginia Academy of Family Physicians, reading:

RESOLVED, that the Medical Society of Virginia support the concept of health care savings accounts as outlined in HR 1080 of the 101st Congress and urge MSV members to contact their respective senators and congressmen in support of this bill, and

RESOLVED, that the Medical Society of Virginia provide pertinent information concerning HR 1080 to its membership to assist them with the composition of correspondence on this matter to their respective senators and congressmen.

2) Also from the VAFP, a resolution on laboratory testing:

RESOLVED, that the Medical Society of Virginia go on record as being opposed to existing federal regulations which require physicians to accept assignment for clinical laboratory services, and

RESOLVED, that the Medical Society of Virginia request the American Medical Association to work for the repeal of federal regulations which require physicians to accept such assignment and support development of alternative regulations making appropriate provisions for laboratories in physician offices.

3) The Report of the Committee on Alternative Delivery Systems.

REFERENCE COMMITTEE THREE: Internal Affairs

Carol S. Shapiro, MD, Chairman

James A. Wassum, MD, Robert T. Mosby, MD, Emerson D. Farley, Jr., MD, Anthony J. Munoz, MD, Charles C. Freed, MD, Robert L. A. Keeley, MD, Harvey D. Smallwood, MD, C. Robert Meloni, MD, William R. Deal, MD, and Sterling Ransome (Student)

DEUNIFICATION

To the members of Reference Committee Three had been given the question of MSV/AMA deunification. Four pertinent documents were theirs for study: 1) the Report of the Society's Committee on Unification/Deunification, advocating reevaluation by the House of its 1985 unification decision; 2) a resolution from the Williamsburg-James City County Medical Society asking for repeal of unification; and 3) the Report of Council, calling for a vote of the membership, with proxy votes as legal tender, on a resolution to deunify. The Council backed up its recommendation by citing the "tremendous loss in membership and income over the past four years of unification" and the overwhelmingly negative comments on unification written into the responses to the 1989 Physicians Opinion Survey (Va Med Oct 1989).

Testimony at Reference Committee Three's hearings bore out and enlarged upon the councilors' concerns. Even Dr. William S. Hotchkiss, the most recent (1987-88) of Virginia's five AMA presidents, had regretfully concluded, he told the Reference Committee, that the Society simply had to deunify, the issue had become too divisive. Meantime, it was bruited about the Richmond Marriott's hallways that the proxy ballots sent out early in October on Council's authorization had brought in an overwhelmingly pro-repeal

vote.

Taking all the material and testimony into account, Reference Committee Three decided to revise the Williamsburg-James City County Medical Society's resolution to read as follows:

RESOLVED, that the House of Delegates determines that the proposed amendment of the Articles of Incorporation of the Medical Society of Virginia to delete the requirement that dues-paying active members must be members of the American Medical Association is of sufficient importance to the Society that it should be submitted to a vote of the voting members of the Society at the meeting to be held on November 11, 1989, and that proxy voting should be permitted at that meeting.

When Chairman Carol Shapiro read out the recommendation to adopt, only one delegate rose with a caveat: Since unification had been adopted by a vote of the House, he said, shouldn't the House make any decision to deunify? Swiftly rejecting this idea, the delegates adopted the resolution as read.

Thereupon Speaker George Broman yielded the podium to Presiding Officer Tom Hulvey, who called to order a meeting of the membership, appointed election inspectors, and ordered the distribution of ballots to those who had not sent in proxies. There were questions from the floor. "Is this entirely legal?" Absolutely, Dr. Hulvey assured the delegates; every step had Counsel Allen



Medical Society of Virginia

1989-1990

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Executive Vice President	James L. Moore, Jr. Emeritus, Robert I. Howard

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Goolsby's seal of approval. "May I change my proxy vote?" You most certainly can, Dr. Hulvey responded, by filling out one of the ballots being circulated and noting on it that it replaces a proxy vote. The ballots were collected, the membership meeting was adjourned, the House of Delegates resumed its deliberations.

Half an hour later the inspectors came back with the count and Dr. Hulvey read out the figures: 2,962 (89%) for deunification, 370 (11%) against.

IMPAIRED PHYSICIANS PROGRAM

Reference Committee Three recommended the adoption of the request from the Physicians' Health and Effectiveness Committee for a budget of \$18,000. The House adopted the recommendation.

Adopted also was Reference Committee Three's recommendation "Dr. William Barney be commended for his pioneer work and leadership with the Physicians' Health and Effectiveness Committee and that the efforts of Dr. David Fluharty, who has made this committee a role model in this country, be applauded". The delegates rose to their feet to commend and applaud. Earlier, Dr. Barney had been elected President of the Medical Society of Virginia 1989-1990 (see page 00).

Dr. Fluharty had introduced a resolution on the AMA's funding of the Impaired Physicians Program, and the Reference Committee recommended its adoption with slight amendments, as follows:

RESOLVED, that the Medical Society of Virginia recommend to the American Medical Association that it continue to adequately fund and maintain an impaired physicians program ("Physicians Assistance Program"), whose charge responsibilities will include, but not be limited to, promoting state medical society impaired physician programs, providing technical assistance to these programs,



The tellers go over the arithmetic of the deunification balloting. Clockwise from left, Dr. Joel R. Poole, Culpeper, Dr. Jack C. Turner, Danville, Dr. William W. Gough, Norfolk, Dr. John W. Knarr, Pulaski, Chairman, and Dr. Monroe G. Baldwin, Lynchburg.

conducting scientific and socioeconomic research, and hosting an annual conference to share research, exchange ideas and provide educational opportunities in the field of physician impairment.

The resolution was adopted as amended.

BYLAWS

Dr. Shapiro reported that Reference Committee Three recommended adoption of the first item in the Supplemental Report of the Bylaws Committee asking that the name of the Public Relations Committee be changed to the "Public Relations and Communications Committee." Instead of adopting, the House elected to refer the recommendation to Council.

A second recommendation from the Bylaws Committee asked for "an extensive revision" of the bylaws. The Reference Committee amended the measure as follows:

RESOLVED, that the Bylaws Committee undertake a comprehensive review of the bylaws of the Medical Society of Virginia in the coming year.

The House of Delegates adopted the recommendation as amended.

BUDGET

The Reference Committee recommended the adoption of the 1989-90 Proposed Budget (see page 00), said the Chairman, and the House adopted it forthwith.

CONSENT CALENDAR

Reference Committee Three recommended the adoption on a consent calendar basis of the Report of the Bylaws Committee and the Report of the Membership Committee. The House adopted both reports.



Fifty-Year Club

For thirty Medical Society of Virginia members, 1989 marked the 50th anniversary of graduation from medical school, thus qualifying them for the 50-Year Club. They were invited to a reception in their honor at the annual meeting, and the eight physicians shown here attended that reception. Seated at center is Dr. Levi W. Hulley, Jr., Richmond. Standing on either side of him are, from left, Dr. Irving Berlin, Hampton; Dr. John H. Thomas, Jr., Greenville; Dr. Saul Kay, Richmond; Dr. William S. Hotchkiss, Chesapeake; and Dr. John Schurr Snider, Irvington. At rear are Dr. J. Warren Montague, Richmond (left), and Dr. Claude Porter Sherman, Martinsville. Here are the new members who were not present for the photograph:

Theodore Bliss III, MD, Norfolk
Beverley B. Clary, MD, Richmond
Jose D. Coll, MD, Richmond
Wayne A. Geib, MD, Albuquerque, New Mexico
Garrett G. Gooch III, MD, Salem
Asa Wesley Graves V, MD, Lacey Spring
T. Ham, MD, Vienna
John C. Hortenstine, MD, Stuart, Florida
William H. Hosfield, MD, West Point
Lawrence A. Jacklin, MD, Falls Church
L. Meredith Johnson, MD, Asheville,
North Carolina

Delmas Bernard Jones, MD, Eureka, California
Robert C. Longan, Jr., MD, Richmond
O. Hunter McClung, Jr., MD, Lexington
Vito J. Murgolo, MD, Silver Spring, Maryland
John L. Patterson, Jr., MD, Richmond
Robert L. Payne, Jr., MD, Norfolk
Nelson Podolnick, MD, Falls Church
Japheth E. Rawls, Jr., MD, Suffolk
Maurice R. Schlanger, MD, Portsmouth
McKelden Smith, MD, Staunton
Lewis Frank Somers, MD, Lynchburg
Mario Stefanini, MD, Richlands

VIRGINIA MEDICAL

Hooray and Hallelujah

UNIFICATION is dead. The ill-fated attempt of the Medical Society of Virginia to require the membership to also become dues-paying members of the American Medical Association has led to wholesale resignation, a membership loss estimated at 1,700 to 2,000. At the 1989 annual meeting, a vote of the Medical Society of Virginia membership favored deunification by a margin of eight to one. (See page 27.) If unification has been the real or major cause of membership loss, the Society will soon regain its former strength. Let us hope this is the case.

The idea behind unification was, and is, a good one.

For greatest effectiveness, medicine must speak with one voice, and the organization best equipped to be that voice is the American Medical Association. The drawbacks are obvious: No one likes to be coerced, and of course, for young practitioners and those in low income specialities, the additional dues can be a burden.

Let us all remember one essential fact: Physicians must resist the divide-and-conquer tactics of government and big business. This can be accomplished only if we work together.

E.L.K., Jr.

Dr. Barney Takes Charge

Following are excerpts from the inaugural remarks of the Medical Society of Virginia's new President, Dr. William H. Barney, before the House of Delegates at the annual meeting.

"The Neurologically Damaged Infant Act has been an anathema to many of our physicians. This is a very disturbing situation to me. We will continue to work to improve this act and to get the assessment removed. . . .

"A committee of great importance this past year is one chaired by Dr. Charles Caravati to develop a better relationship with the Blue Cross/Blue Shield board, and these two groups, MSV and BC/BS, have done just that. In the area of electronic billing alone, negotiations have saved Virginia physicians literally hundreds of thousands of dollars. I have asked that this committee expand its mission to work with other insurance carriers when problems

arise. It will be renamed the Committee on Third-Party Payers and will be chaired by Dr. Latane Ware. . . .

"Perhaps we have not adequately dealt with the increasing problem of illegal drugs and related crimes . . . I believe that our voice can be important, especially in the areas of education and legislation, and I have appointed a new Committee on Substance Abuse, chaired by Dr. Mike Bohan, to study this problem and advise Council and the House of any action that might help. . . .

"I am convinced that we are closer now to national health insurance than we have been in the 45 years that I have been involved in medicine. . . . Let us make certain that our voice is heard through the Federal Legislative Committee under Dr. Norman Edwards working with our congressmen and through the influence of our delegates to the AMA."

PRESIDENTIAL ADDRESS

"We must join hands to find answers."

J. Thomas Hulvey, MD, President 1988-1989,
The Medical Society of Virginia

WHEN I became President of the Medical Society of Virginia, I had a number of things I hoped we could accomplish. We did take many substantial strides forward, but I can't begin to talk about them without acknowledging the people in the MSV organization who have done so much to make those strides possible.

First of all, neither I nor any other MSV President in recent memory could have accomplished anything at all without the unfailing help of our headquarters staff. In particular, my heartfelt thanks to Jim Moore, who is intuitive, has the patience of Job and the very best interests of the Society at heart, and to Ann Gray, who has for years produced an outstanding medical journal on a shoestring and is now making retirement noises. Heaven help us if she really does retire. The MSV staff is small by medical society standards, but they are first rate from top to bottom, and we are very much in their debt.

Now back to what's happened this year—and it's been quite a year. When I started out, I set two major goals—to rebuild the MSV membership and to try to remove the nonobstetric physicians from the annual \$250 assessment. While I didn't reach the final goal on either, in spite of considerable effort, I am very proud to say that we've laid the foundation for both of these goals to be met in the near future. Let me explain.

You don't need to be told the rate at which we've been losing members. The losses this year were the heaviest since the first year of unification. We thought we knew the basic reasons why, but we weren't positive. So we did a survey of all the physicians in the state. I'm sure you've seen the reports of the results in VIRGINIA MEDICAL. They are astounding.

Of approximately 11,000 surveys sent out, over 6,000 completed forms were returned to us. Although many of the responses were critical of some of our policies and of our image, we were greatly encouraged

that so many Virginia physicians cared enough to respond. We've already started to take action on what they told us.

The survey also gave us a lot of additional useful information about what all physicians throughout the state think of The Medical Society of Virginia. We gained invaluable insight into ways we might better serve our members. More importantly, we've learned that we must make a better effort to tell everyone about our programs and solicit their input. We thought that was what we were doing. The survey told us otherwise.

As physicians, we know all too well that we can't treat a disease very effectively until we have established a diagnosis. And so, while I was not able to resolve the membership crisis, I am very pleased to have been President when the diagnosis was found, and we have begun to treat the problem. The real work of implementing what we've learned falls to my very capable successor.

As for Injured Infants Act assessment, we gave it our all and did succeed in getting the General Assembly to authorize a study to improve and broaden the definition of the Act and the financing of the program. We believe that this will soon result in the removal of the assessment and improvement of the overall program as well. Without such a study to use as a basis, there simply was no enthusiasm in the legislature for removing the assessment.

WE'VE done much more in other areas during the year, too. We produced a *Directory of Virginia Physicians* and supported many worthwhile projects, but among the most far-reaching work we've done this year, in terms of providing long-term solutions to a number of problems, is with the state government. Our relationship with our state officials this year has been excellent, and our recommendations have been warmly received by the Governor and the leadership.

A good example is our efforts in the area of indigent care. During my first week in office, I appointed an Indigent Care Committee, which has worked very closely with the Commissioner of Health and the Department of Health and Human Services.

One of the major problems facing this committee is that of indigent mothers who have difficulty finding an obstetrician to deliver their babies. I am certain there is not a physician present who is not concerned about this problem. This committee and the leadership of this Society continue to meet with Commissioner Butterly, with Secretary Teig, and with the leaders of the various specialty societies in an attempt to solve this dreadful crisis. We know we cannot replace those family practitioners and senior obstetricians who have given up obstetrics because of the malpractice crisis and the problems inherent in the practice of rural and intercity obstetrics, and since these problems will not go away on their own, we're looking for other solutions.

Another example of this cooperation with state government is in the area of health care for our senior citizens. They want and need quality health care at an affordable price. In part because of the growing numbers of elderly and resulting costs of providing for their health care, we are now facing a series of challenges that threaten ultimately to replace our health care delivery system with one that may, for the sake of economy, assume the appearance of the Canadian health system and will likely result in the rationing of health care for the first time on a large scale to a select group of patients—our senior citizens.

In addition to our work with state and local government in this area, many of our local medical societies have taken the initiative and are actively working to assure that the elderly receive affordable quality health care in their communities.

We are all painfully aware of today's very important health care issues. We know that there are 26 million uninsured workers in the USA. We know that the

infant mortality rate is far above where it could and should be. We are suddenly aware that the incidence of AIDS amongst our teenagers is skyrocketing, and the problem of drugs in our society exacts a staggering price. These are problems that must be dealt with, and we in the medical community as individual physicians and as a Society must take an active and visible role.

Our survey showed us that our members—and also, I might add, prospective members—look to organized medicine to represent them with government at all levels as these critical issues are addressed. While we in this Society know we have been working hard, we must continue to do so and to draw in our colleagues to help. No one can be allowed to sit idly by and let the government make all of the health care decisions.

It is up to us to see that this does not happen. We have a lot to offer, and we must continue to be available to provide our input and expertise and to demonstrate our resolve to find solutions. I believe that we will be more than welcome, at least at the state level. We must not leave health care planning in the hands of those who are responsible for the postal service, Social Security, and Medicare.

It has been a great honor to serve as President of this Society. I believe that during this past year, in spite of our difficulties, we have established an important link to those physicians in Virginia who do not belong to this Society by asking for their views and their input and by responding visibly and positively. I hope that we will continue to ask periodically for input from members and nonmembers alike.

There is much to do. We must rebuild our membership, put aside our internal differences, and join hands to find answers to the very serious health care problems that face us.

I thank you for your help and cooperation during this past year and for giving me the opportunity to serve you. It has been an exciting and educational experience.

MEETINGS

January 20

Meeting of the Medical Society of Virginia Council, *Richmond*. James L. Moore, Jr., (804) 353-2721.

January 23

Cancer Detection and Prevention (Fairfax Hospital), *Falls Church*. 3.5 credit hrs. Fee, \$40. Cathy Sarraf, 703-698-2572.

January 25-28

24th Annual Meeting of the Neurosurgical Society of the Virginias, the Homestead, *Hot Springs*. Dr. George W. Johnson, 804-320-2758.

January 26

2nd Conference on Comprehensive Care of the Diabetic and Geriatric Foot (Eastern Virginia Medical School), *Virginia Beach*. CME Office, 804-446-6140.

January 27-February 1

Clinical Problems in Gynecologic Surgery (John Hopkins/Emory University/Brown University), *St. Thomas, Virgin Islands*. 20 credit hrs. Fee: \$475. CME Office, 301-955-2959.

January 28-February 2

Gastroenterology Update 1990 (Johns Hopkins/American

College of Gastroenterology), *Park City, Utah*. 20 credit hrs. Fee, \$400. CME Office, 301-955-3839.

January 29-31

How to Get Started in Medical Practice: A Practice Management Workshop for Residents (Southern Medical Association), *Richmond*. 1-800-423-4992.

February 2-4

Annual Meeting of the Virginia Neurological Society, *Williamsburg*. Donna Scott, 804-353-2721.

February 18-25

12th Annual Wilmer Institute Current Concepts in Ophthalmology (Johns Hopkins), *Dorado, Puerto Rico*. 30 credit hrs. Fee, \$475. CME Office, 301-955-2959.

February 23-25

Pediatric Emergency Medicine (Virginia Chapter, American Academy of Pediatrics, and Virginia Pediatric Society), Conference Center, *Williamsburg*. 13 hrs. credit. Judy Suher, (804) 643-8130.

February 25-March 3

4th Annual Innovations in the Diagnosis and Treatment of

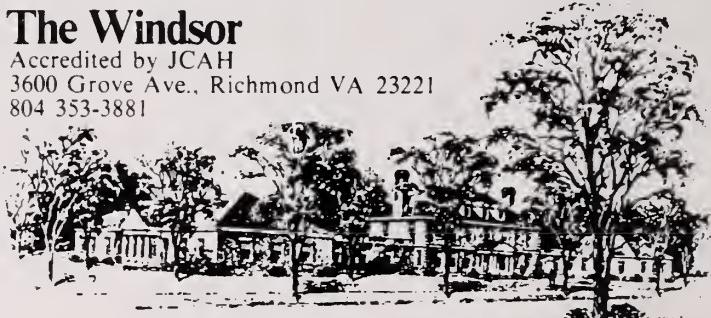


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Manuscript Preparation

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The author is responsible for the accuracy of all statements and references. Acronyms and other abbreviations should be kept to a minimum; unless an acronym is widely known and used by all specialties, it should be fully explained in the text. Refer to pharmaceutical products by their generic names; brand names may follow in parentheses and should carry registered trademarks where applicable. All units of measure should appear in the metric system. References, typed in double-space, should be listed in the order of their citation in the text, not alphabetically. They should follow **VIRGINIA MEDICAL's** typographic style for references; the typist should study this style as it appears in each issue.

Illustrations should be black and white glossy prints, with legends typed in double-space on a separate sheet of paper. **VIRGINIA MEDICAL** has no budget for printing in color; the author who wishes to publish a four-color figure may negotiate to pay for the costs.

Attach to the contribution a covering letter giving the address and telephone number of the person who will correspond about it and address the completed communication to the Editors, **VIRGINIA MEDICAL**, 4205 Dover Road, Richmond VA 23221.

All manuscripts are subject to editorial changes. If extensive revision is deemed necessary, the author will receive for approval a draft of the article as edited.

There are many excellent handbooks of effective writing, among them *The Elements of Style*, by William Strunk, Jr., and E. B. White (MacMillan); *The Careful Writer: A Modern Guide to English Usage*, by Theodore M. Bernstein (Atheneum); and *How to Write and Publish a Scientific Paper*, by Robert A. Day (ISI Press).

Gastrointestinal Disorders (Georgetown University), *Vail, Colorado*. CME Office, 202-687-8735.

March 3

4th Annual Peripheral Vascular Disease Symposium (Fairfax Hospital/Georgetown University), *Falls Church*. 3 credit hrs. Fee, \$40. Cathy Sarraf, 703-698-2572.

March 5-8

Alton D. Brashear Postgraduate Course in Head and Neck Anatomy (Medical College of Virginia/VCU), *Richmond*. Hugo R. Seibel, PhD, 804-786-9624.

March 8-11

Controversies in Obstetrical Ultrasound (Eastern Virginia Medical School), *Snowbird, Utah*. 16 credit hrs. CME Office, 804-446-6140.

March 10-17

7th Annual Wilmer Institute Current Concepts in Ophthalmology (Johns Hopkins), *Vail, Colorado*. 30 credit hrs. Fee, \$475. CME Office, 301-955-2959.

March 11-14

Internal Medicine Seminar (Lloyd Noland Hospital), *Disney World, Lake Buena Vista, Florida*. CME Office, 205-783-5276.

March 14-17

Pediatrics Seminar (Lloyd Noland Hospital), *Disney World, Lake Buena Vista, Florida*. CME Office, 205-783-5276.

March 19-21

Spectrum of Developmental Disabilities XII: Prematurity—Neuro-Developmental Assessment and Intervention Issues (Kennedy Fellows Ass'n/Kennedy Institute Handicapped Children/Society Developmental Pediatrics/Johns Hopkins), *Baltimore*. 20 credit hrs. Juliet M. Nutt, 301-550-9423.

March 23

Sleep Apnea (Eastern Virginia Medical School), *Norfolk*. CME Office, 804-446-6140.

March 24-25

Advanced Cardiac Life Support Provider Course (Fairfax Hospital), *Falls Church*. Fee, \$200. Cathy Sarraf, 703-698-2572.

March 30-April 1

Pediatrics 1990 (American Academy of Pediatrics), *Marco Island, Florida*. Suzanne Goheen, 312-981-7884.

April 6-8

Mammography: Current Concepts and Interpretation (Eastern Virginia Medical School), *Williamsburg*. CME Office, 804-446-6140.

April 6-8

Annual Ophthalmology Conference: Cornea and External Disease (Medical College of Virginia/VCU), *Williamsburg*. CME Office, 804-786-0494.

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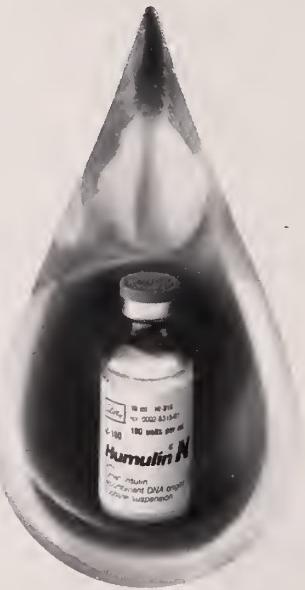
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References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical Letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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New AIDS Law (guidelines and sample patient forms). Va Med October 1989. 2) Physician's Guide to the Natural Death Act, with the form authorized by the 1989 General Assembly. Va Med August 1989. 3) Physician's Kit for the New Virginia High School League Physical Exam (charts, tables, and other clinical material). Va Med September 1989. Send your order to Reprints, Va Med, 4205 Dover Road, Richmond, VA 23221. Be sure to specify which reprint(s) you want.

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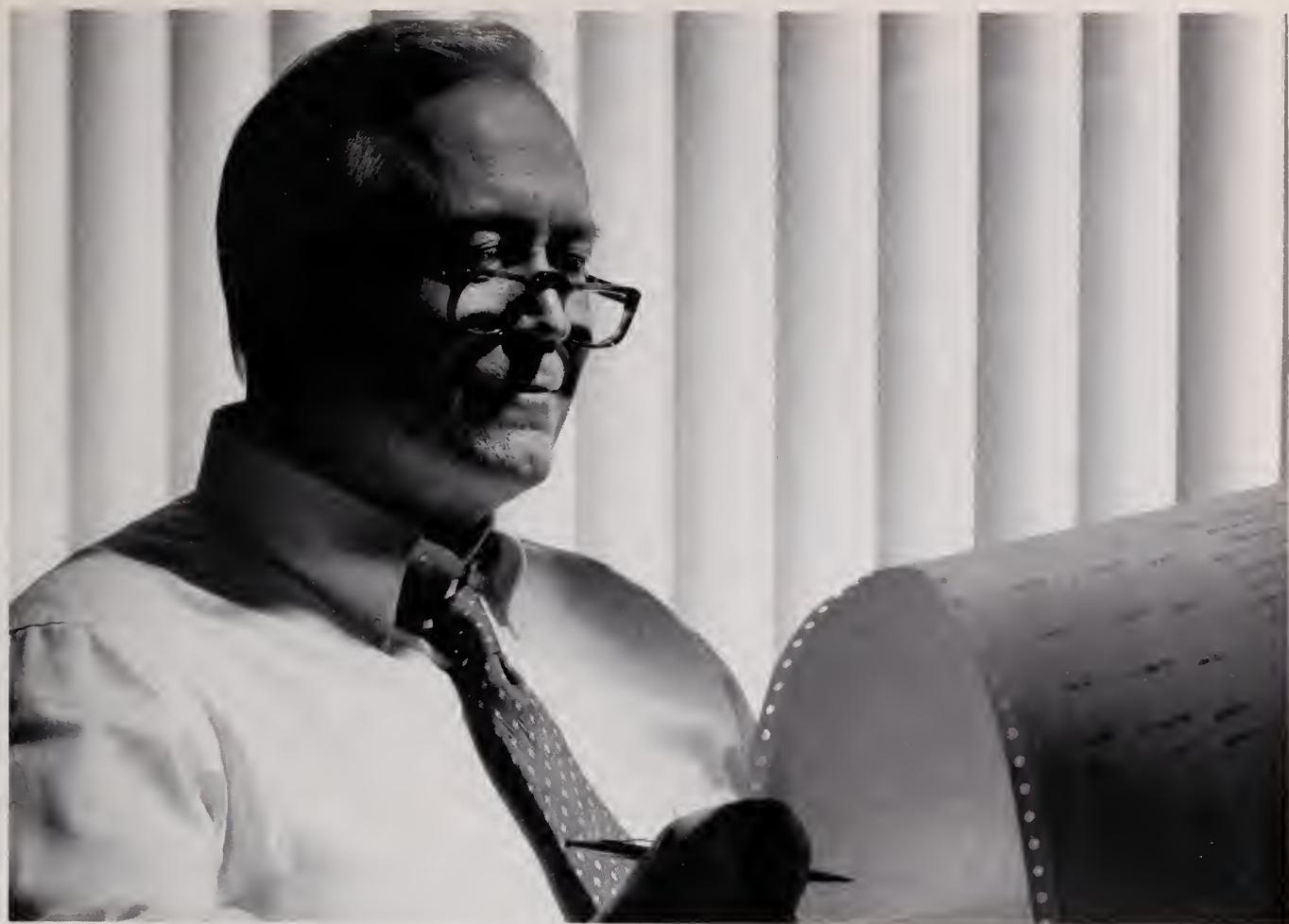
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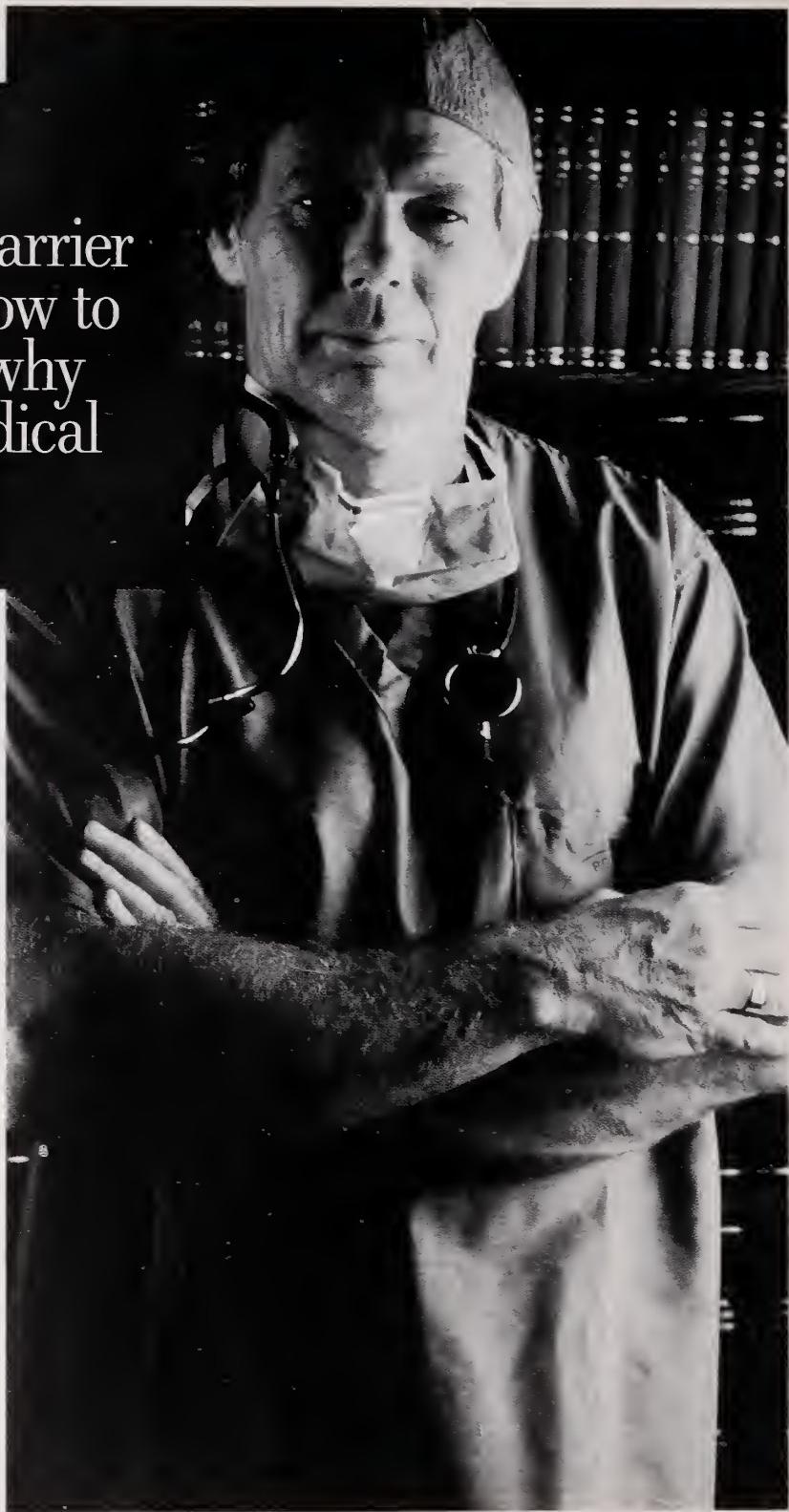
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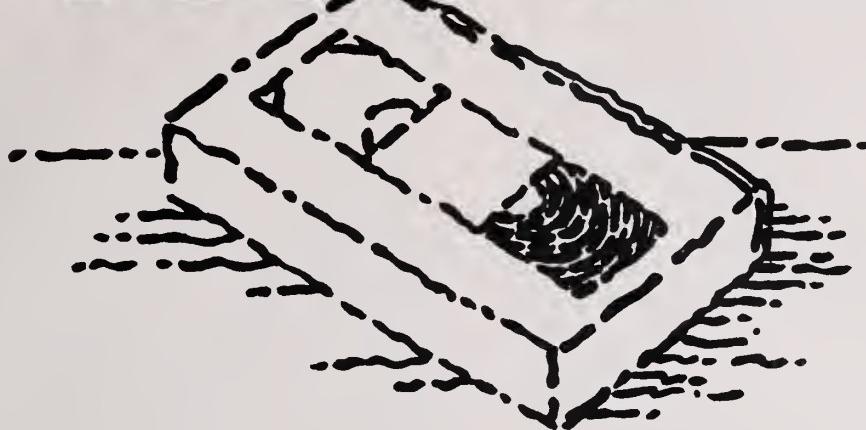


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LETTERS

Taking up the cudgels: cigarettes, cancer and car keys

I feel that the recently published letter of Dr. Stuart Ragland, Jr.,¹ should not go unanswered. He stretches the rules of logic and science to show that the evidence that cigarettes cause lung cancer and other illness is "merely" statistical and that we risk making a "mockery of the profession" for promoting such ideas without rigorous scientific data. This is utter nonsense.

In the first place, even if we had only epidemiological data, those data are very strong. Combining many studies, hundreds of thousands of patients have been followed and a consistent relationship is found between cigarette smoking and lung cancer. Furthermore, the risk increases with the number of years of smoking, number of cigarettes smoked daily, amount of inhalation, and presence of other known carcinogens. The main risk factor for lung cancer in asbestos-exposed workers is not the degree of asbestos exposure but a history of cigarette smoking. The risk of

cancer drops in quitters and is higher than normal in "passive smokers." These facts are hardly "more wrapped in emotion than scientific truth."

In the second place, the only data are *not* epidemiological. Cigarette smoke contains many chemicals which are mutagenic or carcinogenic in bacterial or animal models. Animals forced to inhale cigarette smoke develop lung cancer (although, as in humans, not at a rate of 100%). The fact that not everyone who smokes develops cancer does *not* prove that smoking does not cause cancer. My car doesn't start every time I turn the key. On the other hand, some persons can start a car *without* a key. Do you think for one minute my kid is going to tell me, "But Dad, I didn't start the car; I just turned the key, and we both know that that's not what really makes the car start"? Let's show some common sense here! Volumes of data point to the obvious fact that smoking is one possible step in a complicated path to generating a cancer.

In the third place, he confuses "genetic" with "hereditary" etiologies. (Incidentally, the chromosomal changes he describes are "only statistically associated" with lung cancer and generally have been identified only *after* the cancer has been diagnosed, frequently after the patient has received mutagenic anticancer chemotherapy.) It is possible or even likely that these changes may not be hereditary but induced by carcinogens, e.g., in cigarette smoke.

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In the fourth place, he seems to think that basic research is more important than prevention. Would he have us abandon sanitary water systems now that we can cure dysentery? Stop isolating TB patients now that there is effective chemotherapy? Not obey traffic signals now that we have an efficient trauma system in our state? Why then would he have us continue smoking when there isn't even a cure for the cancer it causes?

To answer his concluding quote with another, I would paraphrase Rene DesCartes, who stated that we should certainly keep an open mind on the issue, but not so open that our brains fall out.

John F. Cary, MD

9590 Surveyor Court
Manassas VA 22110

1. Ragland SRJr. The subject of smoking: more emotion than scientific truth? Va Med 1989;116:451-2

Dr. Ragland replies: Nothing is "silly" about dispassionate scientific enquiry nor is ridicule an acceptable part of a truthful search. Epidemiological studies provide paths that may lead to biological truths. Statistics allow for probabilities. Neither elucidate the mechanisms of disease. They are but adumbrations of basic scientific phenomena. No comment on car keys.

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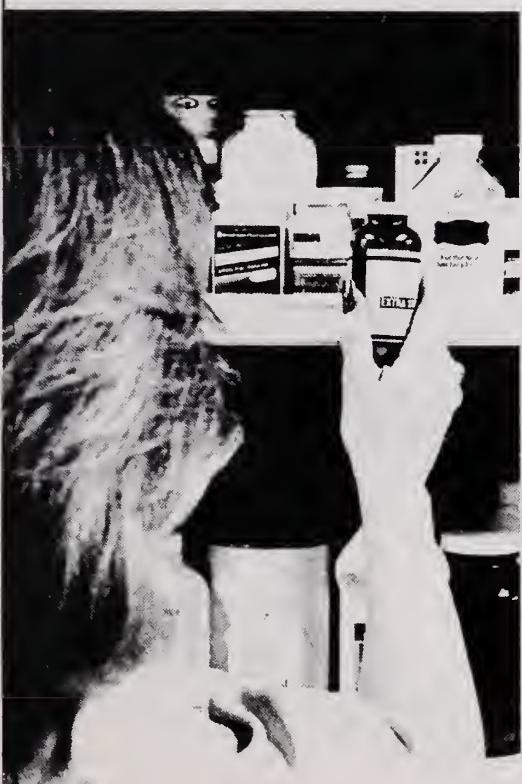
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WHO'S WHO

Two pathologists associated in the management of American Medical Laboratories in Fairfax City, Virginia, have been elected to leadership posts by specialty society peers. They are **Dr. C. Barrie Cook**, who was named president elect of the American Society of Clinical Pathology at its annual meeting in Washington DC, and **Dr. Ira D. Godwin**, who was installed as president of the American College of Nuclear Medicine during its meeting in Chicago. The two men have been associated since 1961, two years after Dr. Cook founded American Medical Laboratories; Dr. Cook is now chairman of the board, Dr. Godwin is president and chief executive officer. Their firm employs nearly 900 persons, making it one of the largest private employers in the Washington metropolitan area. Both men have contributed dynamically to the Medical Society of Virginia. Dr. Cook, a Past President of the Society and winner of its Community Service Award, is currently an MSV delegate to the AMA. Dr. Godwin sits on the Society's Council as the councilor from the 10th District and currently heads up two MSV Committees, Insurance and Professional Liability Review.

Dr. William D. Sasser, Fredericksburg, is the new president of the Virginia Vascular Society. He was elected at the Society's annual meeting in Williamsburg.

New president of the Virginia Society for Pathology is **Dr. Benjamin C. Sturgill**, Charlottesville, who was elected at the Society's annual meeting in December.

New president of the Association of Life Insurance Medical Directors of America is **Dr. Lawrence D. Jones** of Lynchburg, where he is vice pres-

ident and medical director of First Colony Life Insurance Company. Founded in 1889, the Association he heads has a world-wide membership.

To the November 1989 issue's list of three newly elected fellows of the American College of Radiology must be added these two whose names were inadvertently omitted: **Dr. William Bruce Lundein**, Arlington, and **Dr. Brian R. J. Williamson**, Charlottesville.

Dr. Lawrence C. Varner, Farmville, has been named a fellow of the American Academy of Family Physicians.

Dr. Richard P. Keeling, director of the University of Virginia's Department of Student Health, was one of 11 physicians and dentists from over the country honored recently for their care of people with AIDS. The awards were presented by the US Public Health Service. Dr. Keeling, president of the American College Health Association and chairman of the board of the National AIDS Network, was cited for "selfless dedication, compassionate service and outstanding leadership in providing medical care to people with HIV infection and AIDS."

A Norfolk obstetrician/gynecologist who dropped his practice in 1984 and is now "totally addicted to painting" was the winner of the 1989 Urbanna Oyster Festival artwork competition. He is **Dr. William N. Reingold**, whose realistic portrayal of "York River Oystermen" took the prize. Dr. Reingold has won several prizes at shows in the Tidewater area, including the Portsmouth Seawall Show and the Hampton Roads Medley of the Arts. He is represented in Norfolk by Harbour Gallery

and in Virginia Beach by Gallery 32. "I'm the luckiest man in the world," he told a reporter for Urbanna's *Southside Sentinel*, explaining that after 31 satisfying years as an ob/gyn, he has now fulfilled a lifelong ambition to paint full time. "How many other people get two shots at it?" he asked rhetorically. "I don't take it for granted." He is self-taught, paints from photographs, uses acrylic paints, and specializes in scenes on and around the water. His Oyster Festival winner was printed on the cover of the Festival's program and its official T-shirts.

Dr. John A. Jane, Charlottesville, has been elected to a six-year term as a director of the American Board of Neurological Surgery. Dr. Jane has been professor and chairman of neurosurgery at the University of Virginia since 1969.

A geriatrics chair in the Department of Internal Medicine at Eastern Virginia Medical School has been endowed with a \$1 million gift, and it will carry the name of **Professor John Franklin, MD**, who has been a strong supporter of the Norfolk school since its inception and is now director of the EVMS geriatrics program. A graduate of the Yale School of Medicine, Dr. Franklin came to geriatrics by way of internal medicine and cardiology.

Five Medical Society of Virginia members were appointed recently to endowed professorships at the Medical College of Virginia/VCU. They are **Dr. W. Robert Irby**, Charles W. Thomas Distinguished Professor of Rheumatology; **Dr. Austin I. Mehrhof**, Leroy Smith Distinguished Professor of Plastic Surgery; **Dr. Duncan S. Owen, Jr.**, Taliaferro-Scott Distinguished Professor of Internal Medicine; **Dr. John D. Ward**, Harold I. Nemeth Professor of Neurosurgery; and **Dr. Harold F. Young**, Edward and Elizabeth Hirschler Distinguished Professor of Neurosurgery. The appointments brought to 16 the number of MCV faculty endowments.

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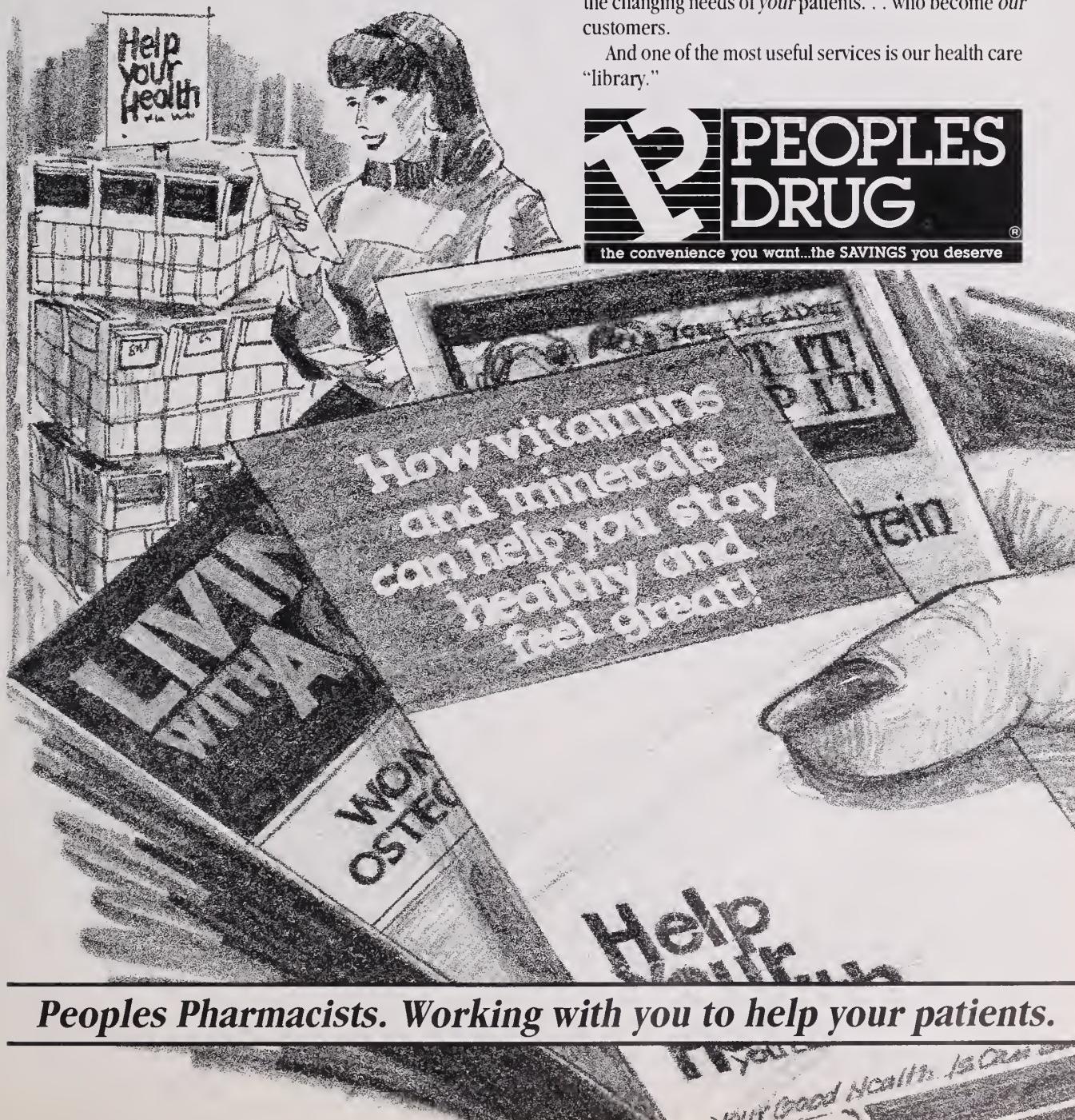
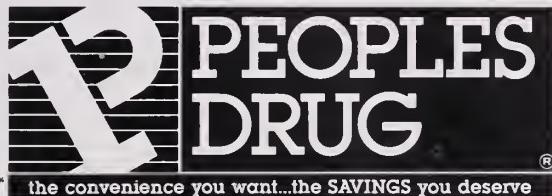
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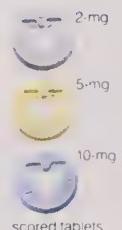
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Medicine Abroad: I. Hospital #3 in Leningrad

By Ray H. Smith, MD, Richmond, Virginia

I HAVE just returned from a tour and medical seminar in the Soviet Union sponsored by the Medical Society of Virginia and other medical societies in the United States. While in Leningrad, I had the opportunity of visiting Hospital #3 with a group of American physicians and here report on that experience.

This hospital was selected for us by Intourist, the national Russian tourist agency which determines what tourists may see throughout the U.S.S.R. Since this particular hospital was chosen, it was our feeling that it was probably one of their better hospitals.

We had a courteous reception at the hospital and a friendly interview through an interpreter with a Russian physician who was the assistant chief physician and "Director of Treatment" of the hospital. We had an informative question and answer session with the physician, and the following consists of what we were told, plus my own observations.

Hospital #3 is a 1200-bed, acute-care, general hospital without obstetric or pediatric services. The hospital is a large, U-shaped, plain, brick building, probably six or eight stories tall, and appeared to occupy four or five acres. It was built seven years ago and is already in poor repair, with broken windows, cracking plaster and in need of painting. There is one small parking lot, which is all that is necessary since very few Russians own cars. A hospital ambulance in the parking lot contained a canvas stretcher of World War II vintage and what appeared to be a first aid box, but no oxygen or other equipment or supplies were evident.

The hospital serves a residential area in Leningrad

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of one million people. About 5% of admissions are referred from clinics and 95% are admitted as emergencies. We did not determine the average daily census but were told that the overall average length of stay is about three weeks. The average length of stay for an acute myocardial infarction is three to four weeks and for an appendectomy and herniorrhaphy, about two weeks. We were also told that the length of stay is similar in hospitals throughout the Soviet Union and that there is no governmental pressure to reduce the length of stay.

The doctor told us that the hospital has a large trauma and active neurosurgical service, but they do not have a CT scanner. Arteriograms also apparently are not done at this hospital. Patients in need of such studies are transferred to another hospital, but it was unclear as to the number and what type of patients are actually transferred.

Pulmonary disease apparently is very common, which is not surprising since most Russians seem to smoke, and the doctor was especially proud of a new arterial blood gas machine. He also was very excited about the recent acquisition of a SMAC machine for blood chemistries, and took great pleasure in showing it to us, although it was not yet in operation.

From what we could observe in an incredibly small laboratory for this size hospital, all laboratory studies were being done manually.

At our request, we were shown the "coronary care unit," which consisted of two rooms, each with four old metal beds, with adequate open space between the two rooms. One room was for males and the other for females. There were no curtains or screens between the beds, and monitors above the beds were not working. The central monitor placed between the two rooms was turned off and when turned on for us,

revealed three monitor strips for eight patients. Two of the strips revealed only straight lines and the other revealed marked 60-cycle interference, making any kind of interpretation impossible. No patient had an IV, and nasal oxygen was administered through a large, red rubber tube. No defibrillator was evident, and hemodynamic monitoring was obviously impossible in this unit. When asked about thrombolytic therapy for an acute myocardial infarction, the doctor did not understand the question. We were told that patients who needed cardiac catheterization or cardiac surgery were transferred to another hospital. We were also told that coronary artery angioplasties were being done in some hospitals but obviously not this one.

On another occasion, we visited a "Preventive

Medicine and Disease Prophylactic Center" for automobile workers, which demonstrated a pretty good picture of medieval medicine being practiced in the late 20th century!

It would appear that medicine in the U.S.S.R. is ten to twenty years behind the U.S., if what we observed is representative of medicine as practiced there.

I would encourage anyone who has the chance to visit the Soviet Union (depending on the political situation), for an unforgettable experience, to see first hand that the Communist system does not work for the benefit of the people and to have a much better appreciation of our country and government despite our problems. I would also advise anyone traveling in Russia not to get sick.

II. Glasnost in Moscow, Tbilisi, Baku

By John W. Hollowell, MD, Portsmouth, Virginia

IN the summer of 1989 I joined a party of 20 American urologists and guests on a two-week tour of Russian medical facilities. The trip was led by Dr. Jay V. Gillenwater, professor and chairman, Department of Urology, University of Virginia. Three of the 15 Soviet republics were visited: Russia, Georgia and Azerbaijan. Visits to medical facilities in these countries were prearranged by Prof. Nicolai Lopatkin, director of the Moscow Urological Research Institute, and his English-speaking associate, Dr. Anatoly Darenkov. An Intourist escort assigned by the Soviet government stayed with the party throughout the trip and was joined at each city visited by a local Intourist guide.

We went first to Moscow, where we stayed at the 30-story Cosmos Hotel, an impressive edifice of several thousand guest rooms. Assignment to room was by coupon issued at the registration desk. This was handed to the "key girl" on each floor in exchange for the room key. The process was reversed on leaving the room each and every time. The constant monitoring provided good security and might well be emulated by American hotels.

Next morning Professor Lopatkin proudly escorted the group through his research laboratory and a portion of the 300-bed Institute. He was reputed to be of high position in the Communist Party, and his station apparently carries many perquisites, among them frequent trips abroad and a good deal of sophisticated equipment. The building was described as ten years

old but looked much older and was of poor construction and finish. The main stairway was a showpiece, with Oriental carpeting over marble steps, but the host laughingly explained that it was built for show and rarely used.

Some mention was made of an organ transplant program, and a busy dialysis unit was visited. The patients were in wards, and sterility precautions were sparse. Catheters draining through dirty rubber tubes into open containers on the floor reflected practices abandoned in this country over 40 years ago. A lithotripter unit (ESWL) was functioning, and the equipment appeared to be a Russian copy of the original West German machine. Dr. Lopatkin answered questions freely, stating that Russian medicine was not up to American standards, pointing out that "the Soviet Union is only 72 years old and the United States has over 200 years of experience. Give us time."

THE next day we made the 1200-mile flight to Tbilisi, capital of the Republic of Georgia and birthplace of Stalin. The semi-tropical, fun-loving atmosphere of Georgia was in marked contrast to Russia. With no notice or explanation (standard practice), Intourist changed our reservations from a modern hotel on the edge of town to the downtown Hotel Tbilisi, an aged but elegant building predating the Revolution. There was no air conditioning, but high ceilings helped relieve the oppressive heat.

Across the street was a blood-stained sidewalk, fronting a government building, where two months earlier 19 students had been slain in a "freedom uprising." At night, students still milled about the

Dr. Hollowell is in the private practice of urology at 226 Fort Lane at Crawford Parkway, Portsmouth VA 23704.



Corner of typical patients' ward in a 1500-bed general hospital in Leningrad. The patient was being treated for an intestinal obstruction. Her surgeon stands at right in the tall hat that is part of the white uniform worn by all physicians in the hospital. *Photograph by the author.*

streets, providing an air of tension, and a loud crash of broken glass that awakened everyone one night proved to come from a champagne bottle hurled through a window by a protesting youth.

In Tbilisi the group had a two-hour visit with Prof. Lauri Managadze, director of the Urological and Nephrological Institute of the Georgian SSR, one of three such institutions in the Soviet Union. He apologized for not inviting us to the Institute but explained that major repairs were being done. Professor Managadze was very open and candid and admitted to a lack of such medical amenities as scans, ultrasound, ESWL. He confirmed information obtained in Moscow that vasectomies are illegal in Soviet countries; abortions, however, are free and anonymous, and the average Soviet woman has eight to ten abortions in a lifetime. The vasectomy ban stems from the government's desire to increase the population above the current 284 million mark (for all 15 republics).

Professor Managadze was asked about the effects to date of perestroika. He was enthusiastic, listing: 1) more openness, democracy; 2) can now send his staff doctors to foreign lands for study without Moscow approval; 3) can now order equipment, such as CT scanners and ESWL, at the republic level without Moscow approval. In other words, decentralization of power has occurred.

FROM Tbilisi we flew to Baku, capital of Azerbaijan, an Asian country with a strong Muslim influence. Baku is an old city on the Caspian Sea, from whence comes 85% of the world's black caviar. A visit to a urology clinic in Baku was surprising: It had good equipment and was well-staffed, possibly because of

its location in an oil-rich, busy, trade city.

A four-hour flight from Baku delivered the tour group to Leningrad, where we stayed at the Hotel Pribaltiyskaya on the Bay of Finland and visited a general hospital of 1,500 beds, 75 of them reserved for urology patients. Medical care in this hospital appeared to be 40-50 years behind the United States. The physical plant was dreary and poor, the equipment antiquated.

HERE are some observations on Soviet health care I brought home with me.

Approximately 70% of Soviet doctors are women, and the percentage used to be larger. Doctors abound in the Soviet system—double the number per population compared with the States. The training is poor. One is required after finishing high school to decide whether to go into academics and what specialty to pursue. Medical school requires six years and is geared to specialization from the start. A lack of residency programs means poor preparation for practice. One female radiologist said her specialty training was limited to six weeks.

There is difficulty getting doctors to serve in rural areas and special incentives are offered. A *feldsher* system abounds. The *feldsher* corresponds to the German "field Barber" and has training comparable to that of a Navy corpsman, or perhaps a midwife. Much rural care is practiced by these paramedics. The government tried to abandon this system in the 1930s but it proved too popular and firmly ingrained to change.

There is a tiered medical system with high party officials receiving good medical care and the common

people getting poor care. The KGB, members of the Academy of Sciences, and Communist Party elite have their own autonomous health systems. Bribes, evident in all of Soviet life, abound for better health services and special care. Unlike England, the Soviet health system has no private or philanthropic enterprise competing with the socialist system.

Only about 3% of the "net material product" of the Soviet Union is dedicated to health care, compared with 12% of the comparable gross national product in the United States. Physician compensation is poor, 250-350 rubles per month. (At the exchange rate then of one ruble = \$1.60, this translated to \$400-\$560,—but early in November I read that the official rate had changed to \$.16 per ruble, one-tenth as much! This reflects the economic chaos of the Soviet Union.) If a physician has a high position in the Party, he/she might receive 500 rubles per month. The chief of surgery in the Leningrad hospital stated he had three incomes but still could not afford a car. His apartment and clothing

are modest. His food is adequate; rarely can he afford to dine out. The nursing profession is held in low esteem and therefore attracts few. Nurses train for two years and are paid 90 rubles per month.

Supervision of health care in the Soviet Union is the responsibility of the Union Minister of Health, who presides over a hierarchy of nearly 6,000 subordinate ministries and health departments in the 15 republics. Much health care is centered around the workplace, with factories building their own hospitals, but they, too, are under the Minister of Health. Again, in this self-described egalitarian society the elite enjoy unusual privilege.

Communist Party aside, one is impressed with the friendliness and sincerity of the people of the Soviet Union. There is a sense of beauty in these people, who through the ages have become tolerant of war and adversity, who accept a life of grief. The United States would do well to continue in its policy of building bridges to friendship and peace.

III. Nit-Picking for the Army in Rabat

By John C. Watson, MD, Alexandria, Virginia

RECENTLY I've been hearing travel advertisements for French Morocco on radio and television. They make me nostalgic for my youth and for Rabat, the capital of Morocco, where the Army sent me in 1943 during World War II. I was in the 45th General Hospital, the Medical College of Virginia unit out of Richmond, technically geared for 1,800 patients. In actual practice overseas, more than one-half of these patients were in tents convalescing.

We sat at Camp Lee for almost nine months, then went overseas immediately after the North African invasion, landed at Casablanca, thence to Rabat, the capital of Morocco. Most of us suffered from the total geographic ignorance common to Americans at that time. We expected lions to jump out of the bushes and found instead a charming French Moroccan capital. The French section surrounded the native section, which was called the Medina, the word meaning "fort." This was a walled-in city with narrow streets and open sewages. Its bazaar had the traditional streets of leather goods, streets of meat, streets of copper, etc., and one U-shaped street, which was the traditional street of prostitution. The Medina, which had typhus, smallpox, and venereal diseases you wouldn't believe, was technically off limits; but the

troops returning, mostly from the Kasserine Pass, which was the disastrous Bull Run of this North African war, paid no attention to the "Off Limits" signs and would follow any native woman into the Medina. The women wore djellabas and held their hoods across their faces; I never saw a veil and it was impossible to tell what color they were unless you looked at their heels in the open-heeled babouches they wore. We were the only medical installation in that area.

The Allied Commander of Rabat was a former lieutenant governor of Michigan who decided that the "manly thing" to do was to legalize whorehouses, police them, and keep them inspected. He ordered our commanding colonel, to supply the medical officer. The colonel did not approve of all this, but he was outranked. Sanitation in the Army table of organization fell to the lab, and I was low man on the laboratory totem pole—not only the junior but the only unmarried officer. You can guess who was assigned the detail.

We took over four of the houses within the Medina, one a house with only native girls. All the girls had those little red tattoos in the middle of their foreheads. One of the houses outside the Medina was named the Papillon Bleu. I submit that only the French would name a whorehouse the Blue Butterfly. Prostitution was licensed in Morocco at that time, and all the girls had cards that looked like drivers' licenses with their

Dr. Watson has retired from the private practice of internal medicine/cardiology. Address correspondence to him at 4826 Maury Lane, Alexandria VA 22304.

pictures, first names, heights, weights, color of eyes, hair, etc. I do not remember surnames. Almost all of them were named Renée, Simone, Adorée, Mimi, etc. Nearly all of them were young and rather pretty. The houses were run by very capable French madams, who looked, dressed, and behaved for all the world like the rather austere French business women on the main street of Rabat. The little prostitutes were carefully trained not to show recognition of any man they might meet on the streets. I used to greet them pleasantly; they were astonished. After all, it wasn't my home town!

Dr. Merlin-Lemas was the health officer of the Pasteur Institute, which was the French name for the Rabat Health Department, was a very pleasant Frenchman with good English and very capable. I liked him immediately. He did all of the exams for venereal diseases and did them much more capably than I could have. My training was in internal medicine with the accent on cardiology, and I had had a year of pathology, and in the Army, if you knew the difference between a laboratory and a lavatory, you ended up in the lab. In Dr. Merlin-Lemas' examinations, which were conducted in the clinic building of the jail, if the girls came up with a disease or were found to have a disease, they were simply jailed until they were cured. Remember that this was before the days of penicillin and the magical drugs. We did have sulfa drugs, however.

Dr. Merlin-Lemas had no interest in body lice. We did because the body louse was the carrier of typhus. It fell to my lot to inspect the girls for lice. This is done by looking for nits attached to the pubic hair. A nit looks like a flake of dandruff and is found by shining a flashlight across the hair and flicking it with a tongue depressor; if it is firmly attached to the shaft of the hair, it is a nit. The hair is then plucked with tweezers and later examined under a microscope.

The girls were paraded in the nude by the madam. I sat on a chair with my flashlight and my tongue depressor, dressed very properly in my overseas uniform with all the tin that the Army gave us to wear. I was about 28 years old, 6' 2 $\frac{1}{2}$ " tall, weighed about 130 pounds, had a doctorate in medicine, and all my specialty training behind me. The girls found this procedure hilarious. I did my very best to keep my dignity and uphold that of the U.S. Army. I did not always succeed.

Another part of my job was to supply the technician who gave prophylactics to all the customers. Each house was limited to U.S. Army personnel only; we had no Navy people. Order was kept by MPs under the command of a very unhappy-looking, very young Second Lieutenant.

I recall the madam of the Papillon Bleu—tall, dignified, about 60, a little gaunt, wore a totally black dress with a tight collar around her neck. My French was minimal, but I knew enough to say, "Bon jour." When



Lieut. John Watson and a native Moroccan in Rabat in 1943. Behind them is the Wall of Medina.
Courtesy of the author.

I finished the inspection, I would say, "Bon soir, Madam, au revoir." She would reply, "Au revoir, mon Capitan" and then add the single English phrase she knew. It had been taught to her by some G.I. wag and was so completely filthy that I cannot write it. She obviously did not know its meaning.

We thoroughly enjoyed Rabat. I would like to see it again.

We shipped out by train for Oran (Algeria) after about nine months and, after staging there, we went on to Naples; and I remained in Italy for the remainder of the war. The Italian war was serious and deadly, and our halcyon days were over.

One more word about Rabat. There was a native hospital run by the French, called L'Hopital Indigenes. The health officer, as a courtesy, asked all of our officers to visit this place, showed us patients with typhus, kalaazar, and a number of other very rare diseases. He took us into an open ward (this was a pleasant, airy building) and showed us a number of sick-looking Arabs, who appeared to have chicken pox. He, I think teasing us, said, "You gentlemen know what these patients have." None of us did. "They all have smallpox." Not a single one of us had ever seen a patient with smallpox. You have never seen a bunch of sophisticated, university doctors so obviously trying to remember the date of their most recent vaccination.

I was overseas $3\frac{1}{2}$ years. I often think that World War II was the only peaceful time in my life. All the rest of my life, I have had to compete like the devil.

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THE current health-care system in the United States is under seige. Gone are the days when physicians accepted a substantial number of charitable patients and covered their expenses by cost-shifting to charges levied upon more affluent patients. Today the insurance companies and business employers, who pay much of the cost of health care, balk at such fiscal manipulations, which result in higher physician fees, hospital costs and insurance premiums.¹

Compounding this trend is the technology explosion, which has resulted in such dramatic improvements in diagnosis and therapy but at an enormous increase in cost. An aging population with increasing numbers of Medicare recipients, at a time when government is cutting back on expenditures in Medicare and Medicaid programs. The impact of litigation, which has resulted in very high liability insurance premiums and, perhaps more importantly, the cost of defensive medicine, which is difficult, if not impossible to quantitate. The AIDS epidemic promises to create enormous financial stresses upon hospitals and physicians in the not too distant future. There is a steadily increasing literature, and concern, expressed by healthcare analysts, on the subject of inappropriate and unnecessary health-care management, which reflects seriously upon the professional ethical standards and self-policing by the medical profession.

Perhaps the most immediate problem is that of the uninsured working poor. This problem has been presented to Congress intermittently since 1974 without adequate resolution, due to congressional preoccupation with an unsolved budget deficit, ethical misconduct among the leadership, pork-barrel politics and the demands of reelection. In the seventies it was estimated that about 25 million Americans lacked insurance. A deep economic recession in the early eighties compounded the problem, and in 1986 it was estimated that 37.1 million people were uninsured in the United States, most of whom were under 65 years of age, approximately 75% of whom were working, and the remainder dependents of employed individuals who were either uninsured or had no dependent coverage. Another 30 million have inadequate insurance or do not perceive a need for it. By the year 2000 it is estimated that 60 million Americans will be uninsured.²

In Virginia approximately one million people are uninsured (17% of the population) whose circumstances parallel those in the national picture. The family incomes of these Virginians reveal that one-quarter have incomes between \$5,000 and \$10,000 annually; 40% receive \$10,000-25,000 annually; and 21% earn \$25,000 or more, many of whom perceive no need for insurance.³

The impact of such lack of coverage upon Virginia's health-care delivery system was estimated in 1986 to cost approximately \$300 million, of which about one-third was borne by private hospitals but does not include the charitable contributions made by the practicing physicians within the state.³ This is due in large part to the limited access to adequate primary care and preventive medicine by the uninsured, resulting in a disproportionate number of severely ill patients requiring hospitalization and greater than average therapy. A survey by the Virginia Hospital Association in 1988 revealed that 86% of the uninsured admissions were of an acute, emergency nature, as compared with 37% of insured admissions. Forty per cent of these patients were unable to obtain adequate outpatient care prior to admission, and it is estimated that 26% of admissions could have been prevented with adequate primary care.³

The impact of inadequate primary medical care is noted, particularly in those suffering from chronic, debilitating diseases. Such individuals constituted one-third of the emergency admissions and showed an admission rate 25 times that of more affluent and insured citizens.³

The most distressing aspect of this uninsured status is the effect upon the children. This involves 32% of the uninsured, or 11 million children in the country. Inadequate prenatal care leads to the high infant mortality rate, as compared with other democratic countries, and this is stressed by those who advocate a national health policy. Lack of well baby care, vaccination and primary pediatric care often result in dire consequences for the individual and society at large.

What should be done about it? Sen. Edward M. Kennedy has proposed a bill that would require all employers to provide health insurance coverage to their employees, thus extending coverage to an estimated two-thirds of the uninsured. Karen Davis and

Gary Robbins testified in 1987 before the Committee on Labor and Human Resources of the U.S. Senate and expressed the opinion that such a policy would cost between \$23-100 billion and would result in the loss of 120,000 to one million jobs.⁴ The Massachusetts experience would tend to support this assertion.⁵

The Virginia Indigent Health Care Trust Fund derives \$8.9 million from the state's General Fund, and approximately \$6.1 million from hospitals that provide charitable care below a given standard. It is estimated that this will provide only 6% of a projected \$250 million cost of charitable care for the uninsured. This is only a token attack upon the problem and an attempt to resolve a societal problem at the expense of a very small segment of Virginia's industrial complex, i.e., the hospitals themselves, who are already providing significant charitable care.

Blue Cross and Blue Shield of Virginia is attempting to develop a low-cost insurance benefit program which should prove attractive to small business and families with limited funds. To be effective and affordable, the state-mandated benefits currently in force must be removed from this coverage.

Clearly, such efforts to resolve the problem are highly commendable but merely nibble at the edges. The size of the problem demands a more aggressive, cooperative effort between government, industry, insurance carriers, the medical field and the public. It is outrageous that millions of young American women lack prenatal care, which would cost far less than one Stealth bomber, or a fraction of our foreign aid. Eleven million children have little or no access to adequate pediatric care, while \$180 billion is spent annually to support the presence of American troops, tanks and weapons in a Europe which has now recovered from World War II and has a population and a GNP at least as large as ours.

Virginia, in spite of its outstanding economic growth, which is one of the best in the Nation, is now only 42nd in its Medicaid coverage. The state's increased prosperity has resulted in the reduction of federal funds for Medicaid, while the threshold for Medicaid eligibility fell from 76% of the federal poverty level in 1975 to 39.5% in 1986. The federal non-farm poverty level for a family of four is approximately \$12,000; a comparable family in Virginia would not be eligible for Medicaid if it earned \$5,000 or more.

The State of Massachusetts enacted legislation in 1988 which required employers with more than five employees either to underwrite their health care premiums or pay a tax of 12% on the first \$14,000 of wages to create a risk pool for those not otherwise covered. This act was passed during a period of relative prosperity, but more recent revenue shortfalls in the state have raised serious questions as to its viability.⁵ Nevertheless, numerous states have viewed the Massachusetts action with considerable interest.⁶

More recently representatives from the AMA testi-

fied before Congress and recommended a pluralistic approach involving the private sector, the states, and expansion of Medicaid coverage in an attempt to deflect Congress from a comprehensive system.⁷

In 1986 a group of concerned citizens formed a nongovernmental National Leadership Commission on Health Care to assess the major problems in health care, namely, cost, quality and access to care. Their deliberations resulted in a report enunciating a proposal not unlike that proposed by the AMA, which would preserve the finest medical system in the world and ensure its availability to all Americans.⁸

I am convinced that we are rapidly approaching the crossroads. Do we accept the responsibility and active involvement in a program such as proposed above or are we prepared to do nothing and accept a comprehensive health plan similar to that in Great Britain or Canada?

We physicians still enjoy considerable prestige and political influence disproportionate to our numbers. If we are to continue to advocate the high traditional standards of our profession, then we must unite through our local, state and national medical societies and actively engage in the political process to ensure its survival.

The decision is ours. Quo vadis, Medicine?

DENNIS A. J. MOREY, MD

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Dr. Morey retired recently from his practice of gastroenterology and is now legislative representative for Henrico Doctors Hospital, Richmond. Address correspondence to him at 2870 Braidwood Road, Richmond VA 23225.

Eosinophilia, Myalgia Associated with L-Tryptophan Use: Case Report

Daniel M. Becker, MD,
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Stephen M. Smith, MD,
Charlottesville, Virginia

ON November 11, 1989, the Food and Drug Administration issued a warning that L-tryptophan use may be associated with eosinophilia and severe myalgia after 30 reports of this syndrome had been reported from six western states.^{1,2} Since we could find no detailed clinical descriptions of such patients, including their responses to corticosteroids, we here share our experience, beginning in September 1989, in the treatment of a patient with eosinophilia and myalgia who, it turned out, had been using L-tryptophan.

Case Report

A 42-year-old white female previously in excellent health presented on September 6, 1989, with a 4-day history of fever, myalgia and arthralgia after vacationing on eastern Long Island. There were no respiratory or dermatological symptoms. She and her husband had noticed swollen eyes during the first few days of this illness. She denied tick bites, although tick exposure was likely. She had taken a single dose of an oral penicillin for her symptoms, and the only other medication was L-tryptophan (2 grams QHS), which she had been using each night since July 1989 to help her sleep. She obtained this medication without prescription at a local health food store. On physical examination in the office she was afebrile. There was no lymphadenopathy, rash, synovitis, muscle weakness or tenderness. Laboratory results for this visit and others are summarized in Table 1. On September 8 she felt worse and had developed mild orthopnea but no cough. On examination she had faint bibasilar rales and lymphadenopathy. Small (<0.5 cm), nontender, moveable cervical, supraclavicular, axillary and epitrochlear lymph nodes were felt. Her chest x-ray (Fig. 1) showed interstitial pneumonitis with bilateral blunting of the costophrenic angles.

The total white count

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Submitted 11-29-89.

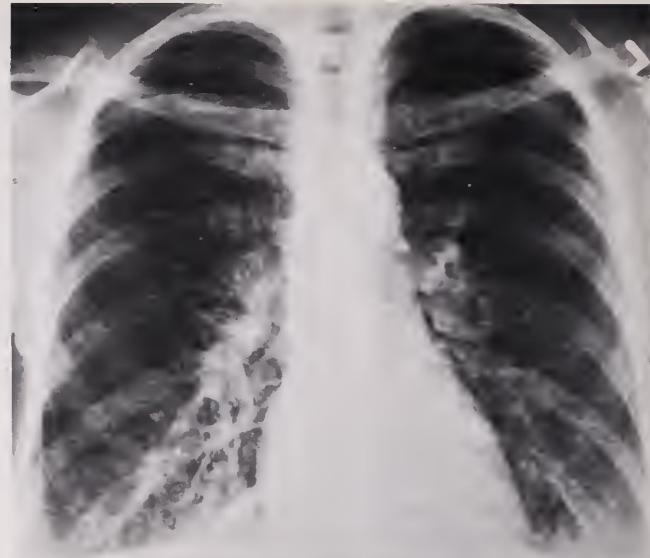


Fig. 1. Chest x-ray showing bilateral interstitial pneumonitis with costophrenic blunting.

was 10.1 K/uL with distinct eosinophilia (15%). Erythromycin (250 mg po QID) was given for 7 days, and while her chest x-ray cleared, she remained ill. On September 14 her transaminases were measured and noted to be elevated.

She was admitted to the University of Virginia Hospital from September 22 through 26 for detailed evaluation. At this time her predominant symptoms were myalgia and arthralgia. She was afebrile in the hospital, and had a transient (one night) pruritic erythematous rash. The lymphadenopathy was still present. There was no synovitis, weakness or muscle tenderness. Marked eosinophilia persisted (Table 1). Her transaminase determinations were lower. The erythrocyte sedimentation rate was elevated, but rheumatoid factor and antinuclear antibody (mouse and human substrate) were not detectable. Total complement was normal, and the anti-C1q assay for immune complexes was also normal. Fasting cortisol was normal, and her PPD skin test was negative with a reactive anergy panel. A *Borrelia burgdorferi* titer (measured by the Mayo Clinic laboratory) was less than 250 antibody response units and considered negative for Lyme disease. There was no evidence for parasite infestation: Stool for ova and parasites was negative repeatedly; toxocara and trichinella antibodies were negative; a string test of duodenal contents was negative for strongyloides and other parasites; total IgE was normal. Visceral larva migrans was nevertheless the leading diagnosis, and she was given thiabendazole (500 mg BID for 3 days). This treatment did not elicit a pruritic response. Use of nonsteroidal antiinflammatory agents provided minimal relief of muscle and joint pain. She was given lorazepam (0.5 mg) in lieu of L-tryptophan for sleep.

At this point in the illness there was still no diagnosis. The patient remained almost totally disabled with

Table 1. Clinical and Laboratory Findings.

Month Day	September					October			November	
	6	8	14	22	25	2	10	19	14	22
<i>Clinical Features</i>										
Temperature (°C)	38.2		Afebrile after September 14							
Weight (kg)	52.5			51.0		49.7		46.6		48.5
Lymphadenopathy	None	↑	↑	↓		slight		none		>
Myalgia	Throughout	—	—	—						
Arthralgia	Throughout	—	—	—						
<i>Laboratory Results</i>										
Hemoglobin (g/dL)	13.3	11.6	13.0	12.7	12.4	12.1	13.4	14.0	12.9	11.6
Hematocrit (%)	40.0	35.0	40.0	39.0	37.6	37.0	41.0	43.0	39.2	35.8
WBC (K/μL)	9.3	10.1	21.2	21.0	17.7	12.6	11.8	16.5	6.5	6.7
Eosinophils (% total WBC)	8	15	29	56	55	40	0	2	0	4
ESR* (mm/h)				66		12				
AST† (U/L, normal 7-40)			103	52		39	17			
ALT† (U/L, normal 6-65)			163	95		60	53			
LDH (U/L, normal 230-450)				748		659	379			
CK (U/L, normal 30-200)					15		17			
Aldolase (U/L, normal 2.5-14.0)							4.8			

* ESR-erythrocyte sedimentation rate

† transaminases: AST-SGOT, ALT-SGPT

myalgia, arthralgia and fatigue, and the eosinophil count remained perplexingly elevated. Largely to exclude lymphoma, a left axillary lymph node was excised on October 4. The histopathology was negative for lymphoma or granuloma, and all cultures (mycobacteria and fungal) have been negative. Symptomatic treatment with prednisone (20 mg BID) was initiated on October 6 for what seemed to be serum-sickness with an unknown precipitant. The patient's symptoms improved within 72 hours of beginning corticosteroids. As expected, the eosinophilia disappeared with initiation of prednisone. Within a week the patient stated she was "90% improved" in terms of muscle and joint pain. Because of persistent muscle pain and subjective muscle weakness, an EMG was performed on October 12. There was no evidence of myositis or neuropathy on this study. She developed muscle cramping at the time prednisone was started, and this symptom seemed to respond to quinine sulfate (200 mg BID). With the clinical improvement and the lack of evidence for a systemic vasculitis, the corticosteroids were tapered relatively quickly, and her last dose of prednisone was on November 12. The same day the local newspaper printed an Associated Press story linking L-tryptophan to an illness identical to that described in this report.¹ The patient read the story, made the diagnosis, and informed her physicians. At the time this was written the patient had been off corticosteroids for 10 days and had continued to improve. Her eosinophilia did not reappear.

Discussion

In this case what seemed initially to be a viral syndrome evolved into a 10-week diagnostic dilemma with pneumonitis, lymphadenopathy, hepatitis, myalgia, arthralgia and persistent eosinophilia. Investigations into the cause of this illness focused on the laboratory finding of eosinophilia. As listed in Table 2, there are many diverse causes of eosinophilia.³⁻⁵ While

many of these could be excluded on clinical grounds, it seemed necessary to search compulsively for helminthic infestation, to look for serological evidence of a systemic vasculitis, and to biopsy an enlarged lymph node when persistent symptoms made lymphoma an ominous possibility. Despite the wide range of diagnostic possibilities, the clinical picture did not clearly match any of the described causes of eosinophilia. While the patient had hypereosinophilia, she did not have it long enough for either a diagnosis of idiopathic hypereosinophilic syndrome or the more morbid end-organ manifestations (cardiac, CNS) of this entity.^{5,6}

Once the link between L-tryptophan and eosinophilia was recognized, the diagnosis in this case became obvious. Our patient fits the case definition proposed by the Centers for Disease Control.² While eosinophilia and myositis have been reported previously, this association has been rare and usually noted as a manifestation of idiopathic hypereosinophilia syndrome^{7,8} or as a feature of focal myositis.⁹ The triad of eosinophilia, myositis and pneumonitis has been reported but is also rare.¹⁰

The pathogenesis of L-tryptophan associated eosin-

Table 2. Disorders Associated with Eosinophilia

Allergy or hypersensitivity	Neoplasia
Asthma	Hodgkin's disease
Seasonal rhinitis	Mycosis fungoides
Drug reactions	Acute lymphoblastic leukemia
Parasitic disease	Sezary syndrome
Strongyloidiasis	Solid tumors
Trichinosis	Cutaneous disease
Schistosomiasis	Allergy
Toxocara	Dermatitis herpetiformis
Filariasis	Immunodeficiency disease
Connective tissue disease	Wiscott-Aldrich syndrome
Necrotizing vasculitis	Hyper-IgE with infection
Eosinophilia with fasciitis	IgA deficiency
Rheumatoid arthritis	Endocrine disease
Systemic lupus erythematosus	Adrenal insufficiency
Idiopathic hypereosinophilia syndrome	Pituitary insufficiency

ophilia and myalgia is not known. The widely reported toxic-oil outbreak in Spain in 1981 established precedent for a toxin-induced neuromuscular illness with eosinophilia, hepatitis, pneumonitis, and weight loss.¹¹ To date there is not enough clinical information to anticipate the natural history of L-tryptophan-associated eosinophilia and myalgia. While our patient has improved significantly with corticosteroids and withdrawal of L-tryptophan, she still suffers from fatigue and muscle pain. In the toxic-oil outbreak, prolonged neuromuscular symptoms were not uncommon.

The CDC learned of 154 cases within the first few days of its initial report.² We have learned of three other patients with this syndrome in the past week. Undoubtedly, physicians in practice will encounter patients with disabling myalgia and arthralgia. Eosinophilia should be sought and the medication history reviewed carefully. Corticosteroids should be considered once L-tryptophan use and eosinophilia are noted. Because the symptoms are frequently disabling, treatment should not be delayed pending an exhaustive evaluation of the myriad causes of eosinophilia. However, since some infections that cause eosinophilia are made worse by corticosteroids (e.g., tuberculosis, strongyloidiasis), the initial evaluation should include PPD skin testing and stool examination for parasites. As physicians in practice become familiar with this new syndrome, early diagnosis and treat-

ment can replace the painful odyssey of our patient.

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ABSTRACTS

The following abstracts are to be presented at the regional meeting of the Virginia Chapter, American College of Physicians, and the Virginia Society of Internal Medicine on March 24 in Richmond. Dr. Donald MacLean Switz is program chairman.

Toxic Shock Syndrome with Acute Renal Failure Secondary to Group A Beta-Hemolytic Streptococcal Exudative Pharyngitis. David J. Thaler, DO, and Joseph Snyder, MD, Portsmouth.

The toxic shock syndrome has been shown to result from exposure to bacterial exotoxins, and, previously, from infection with *Staphylococcus aureus*. A cluster of cases has recently been reported in the western United States involving group A beta-hemolytic streptococcal skin or musculoskeletal infections including cellulitis, infected surgical wounds and suppurative thrombo-phlebitis, as well as peritoneal and pharyngeal infection. The cases of pharyngitis all involved other concurrent sites of infection, including endophthalmitis and myositis.

We report a case of toxic shock syndrome secondary to culture-proven group A beta-hemolytic streptococcal exudative pharyngitis. The case occurred in eastern Virginia and the patient had no significant travel history. The patient presented with all the classical symptoms of toxic shock syndrome, including hemodynamic instability, rash, hypocalcemia, hypokalemia and renal and liver dysfunction. Cultures of the blood and urine failed to yield an organism; however, the patient had a marked exudative pharyngitis which yielded group A beta-hemolytic streptococcus by routine culture. No infected sites could be identified. Renal dysfunction was not associated with a nephritic sediment as would be expected in a post-streptococcal glomerulonephritis. The complements were all within normal limits. This case may demonstrate a new pattern and association for toxic shock syndrome and must be considered now in patients presenting with group A beta-hemolytic streptococcal pharyngitis and renal failure.

Acquired Toxic Methemoglobinemia and Pulse Oximetry: Clues to Diagnosis in a Paradoxical Resuscitation Puzzle. Alan Rolfe, MD, Portsmouth.

Pulse oximetry is a simple noninvasive method for continuous monitoring of hemoglobin (Hb) saturation, and is an indirect measure of arterial PAO₂. Although this form of monitoring is now a standard of care in intensive care settings and for patients undergoing surgical anesthesia, endoscopy, bronchoscopy, and mechanical ventilation, internists and subspecialists who use pulse oximetry monitoring should be aware of both its advantages and its limitations.

Acquired toxic methemoglobinemia (ATM) with se-

vere cyanosis is potentially fatal but is easily curable if it is correctly diagnosed. In this condition, the physical appearance, pulse oximetry saturation, arterial PAO₂, and calculated Hb saturation present a paradox. A 24-year-old white male developed cyanosis after elective intubation and anesthetic induction. Pulse oximetry indicated Hb saturation levels of 82%. Usual resuscitation measures failed to reverse the patient's cyanosis despite documented arterial PAO₂ of 564 torr and 100% calculated Hb saturation on 100% FIO₂. Hb analysis revealed 26% methemoglobin which was treated with intravenous methylene blue. Topical benzocaine used for local anesthesia prior to nasotracheal intubation was retrospectively identified as the cause.

A review of case reports in the literature of ATM revealed significant delays in diagnosis; delays averaged 2 hours and exposed many patients to intensive and hazardous resuscitation procedures. Low pulse oximeter saturations should alert physicians to the presence of unsuspected methemoglobinemia in the appropriate clinical setting. A brief discussion of pulse oximetry is presented. The pathophysiology underlying this resuscitation puzzle and the basis of treatment of ATM are reviewed.

Modern Day Pellagra in South Hampton Roads. Joseph Czerkawski, MD, and Herschel Estep, MD, Norfolk.

Classical pellagra once endemic in Southern U.S. is rarely diagnosed nowadays. After seeing a case of pellagra, we wondered about occurrence of this disorder in modern times. We examined the records of cases of niacin deficiency diagnosed in South Hampton Roads hospitals between years 1981 through 1989. Only nine cases of pellagra were recorded during this time, 4 cases showing all classic manifestations, and 5 cases having features of niacin deficiency excluding rash. Compared to a series when pellagra was endemic (1912-1944), we report that the average age is now older (60 vs 40 years), mental status changes are much more frequent (100% vs 26%), but the classic skin manifestations are seen in only 45% of our patients versus 85% from prior series. Alcoholics made up our entire population, which was not true in the earlier series. Both series established the diagnosis of pellegra on the basis of clinical manifestations, as biochemical criteria were not helpful in establishing the diagnosis. Furthermore, it is likely that patients in our series received suboptimal doses of nicotinamide.

Conclusion: We suggest that classic manifestations

of modern day pellagra may be present with niacin deficiency. However, the disorder should be considered in alcoholic patients with mental status changes with or without other commonly accepted classical manifestations.

Intramural Hematoma of the Gastrointestinal Tract: Case Report and Review of the Literature. Michael P. Jones, MD, *Richmond.*

Intramural hematomas of the gastrointestinal tract are uncommon. Fewer than 350 cases have been reported to date. Two-thirds of these are related to blunt abdominal trauma, but an increasing number of cases are seen in patients receiving anticoagulants. We present the case of a patient with a spontaneous jejunal hematoma secondary to coumadin intoxication.

A 66-year-old male taking coumadin for recurrent thromboembolic disease presented with the sudden onset of abdominal pain, nausea and vomiting. There was no history of trauma. Abdominal examination revealed diffuse tenderness and guarding without mass or peritoneal signs. NG tube aspirate and rectal examination revealed no evidence of bleeding. There was no clinical or laboratory evidence of biliary or pancreatic disease. The prothrombin time was increased to 38 seconds. CT scan of the abdomen revealed a large hematoma involving the proximal jejunum with extension into the mesentery. The patient was managed supportively with NG tube decompression, intravenous fluids and correction of his coagulopathy. His condition rapidly improved over the next several days and he was discharged. An upper gastrointestinal series obtained one month after discharge revealed resolution of the lesion.

A review of intramural hematomas of the gastrointestinal tract with respect to etiology, clinical and radiographic features and management will be presented.

Inhibition of Cardiac Allograft Atherosclerosis by Dehydroepiandrosterone. D. M. Eich, MD, D. E. Johnson, MD, J. E. Nestler, MD, D. Ko, MD, M. L. Hess, MD, and A. S. Wechsler, MD, *Richmond.*

Accelerated atherosclerosis (CAD) is a major cause of morbidity and mortality in cardiac transplantation. Dehydroepiandrosterone (DHEA) is an endogenously produced hormone which has been shown to decrease atherosclerosis in animals. We examined whether DHEA could inhibit CAD using outbred rabbits with heterotopically transplanted hearts. Accelerated atherosclerosis was produced by feeding these animals a 1% cholesterol diet. The animals were immunosuppressed with cyclosporine (5 mg/kg) and azathioprine (1 mg/kg), and they were randomly divided into 4 treatment groups: 1) low cholesterol diet, n = 5, 2) 1% cholesterol n = 5; 3) low cholesterol 0.5% DHEA, n = 5, and 4) 1% cholesterol + 0.5 DHEA, n = 7. The animals were sacrificed at 5 weeks, and the hearts

were perfused at systemic pressure. The hearts were fixed in 10% formalin, the entire coronary arterial tree was cross-sectioned and microscopic slides were prepared. Morphometry was performed on the arterial sections (n = 840) and the percent cross sectional luminal area stenosis was calculated. The mean percent stenosis was 32% in Group 2 and 9% in Group 4. Thus the coronary arteries of the Group 4 animals showed a 72% decrease in luminal stenosis as compared to Group 2 ($p < .01$). There was no significant atherosclerosis in Groups 1 and 3. We conclude: DHEA inhibits accelerated atherosclerosis in this transplant model.

Adrenal Insufficiency Secondary to Disseminated Cryptococcus Neoformans in a Nonimmunocompromised Host. Sharon M. Messics, MD, and Richard K. Sterling, MD, *Richmond.*

Destruction of the adrenal cortex, regardless of the nature of the underlying process, leads to primary adrenal insufficiency. The leading causes of primary adrenal insufficiency are autoimmune disorders and tuberculosis. Other less common causes include neoplasm, hemorrhage, amyloidosis, and fungal infections. We present a case of disseminated *Cryptococcus neoformans* as a cause of primary adrenal insufficiency in a nonimmunocompromised host, without evidence of pulmonary infection nor obvious exposure to the organism. Diagnosis of adrenal insufficiency was made by appropriate ACTH and cortisol levels, and histology of adrenal tissue, obtained by computed tomographic (CT)-guided needle aspiration, revealed the presence of *Cryptococcus neoformans*. To the best of our knowledge, this is the fourth reported antemortem biochemically and histopathologically proven case of cryptococcal-induced adrenal insufficiency.

Effect of Residual Antegrade Coronary Flow on LV Function, Myocardial Perfusion, and Survival after Uncomplicated Myocardial Infarction. James D. Berigan, MD, and Robert S. Gibson, MD, *Charlottesville.*

The effect of residual antegrade coronary flow on LV function and perfusion (at 10 d and 3 mos), and on mortality during 43 ± 18 mos of follow up was examined in 190 consecutive pts (age ≤ 65 yrs) with MBCK confirmed acute uncomplicated MI. All pts were prospectively evaluated with cardiac cath, 24 hr Holter, rest radionuclide ventriculography (RVG) and either exercise (GXT, n = 179 or rest (in = 11) TI-201 scintigraphy within 3 d of hospital discharge (11 ± 3 d). RVG and TI-201 studies were repeated at 3 mo. No pt received thrombolytic therapy, or underwent PTCA or CABG prior to the 3 mo studies. For purposes of analysis, pts were grouped according to the presence (G1, n = 64 [34%]) or absence (G2, n = 126) of TIMI grade 2-3 flow in the infarct related artery (IRA). There were no differences between the 2 groups in age,

prevalence of prior MI or angina, peak CK (measured q4 hrs), Killip class in the CCU, or Lown grade \geq 3 arrhythmias at 8 ± 3 d. Also, G1-2 pts had similar 10 d/3 mo GXT findings (exercise duration, peak RPP or workload achieved, angina and/or ST seg depression), and TI-201 evidence of increased lung uptake and multivessel disease (MVD). However, G1 pts did have lower angio Jeopardy scores ($p < .05$) and less angio evidence of MVD ($p < .01$). G1 pts with LAD infarcts had higher LVEFs on both the 10 d and 3 mo RVG study (48% vs 39% and 48% vs 40%; $p < .05$), but no such difference was noted between G1-2 pts with RCA or LCX infarcts. Further, no difference in LV size was found in any G1-2 subgroup at 10 d or 3 mos. TI-201 studies at 10 d and 3 mo revealed fewer scan segs with persistent defects ($p = .04$ and $p = .03$) and more scan segs with redistribution ($p = .003$ and $p = .036$) in G1 pts. Kaplan-Meier life table analysis showed similar reinfarction rates between G1-2; however, survival was better (.93 vs .81, $p = .015$ [Breslow]) among G1 pts. Significant independent predictors of survival (Cox regression analysis [86 variables]) included smaller infarct size by TI-201 and RVG criteria, improvement in infarct zone asynergy between 10 d and 3 mo, absence of reinfarction, and a patent IRA.

Thus residual antegrade coronary flow is associated with less TI-201 evidence of myonecrosis, better LV function in LAD infarcts, and is an independent predictor of improved long term survival.

Secondary Gastric Syphilis in a Human Immunodeficiency Virus Type I (HIV-I) Coinfected. Dorothy C. Garner, MD, J. Boyd Francis, MD, and Charles J. Schleupner, MD, Roanoke.

The natural history and response to therapy of syphilis may be altered by coinfection with HIV-I. In patients with underlying HIV-I infection, syphilis has been documented to progress from initial infection to neurosyphilis in less than 6 months. Furthermore, progression to neurosyphilis has been documented in HIV-I coinfecting patients despite standard treatment with intramuscular benzathine penicillin.

We report a 27-year-old HIV-I antibody-positive black male with a radiographically demonstrated antral ulcer which failed to respond to conventional treatment. Esophagogastroduodenoscopy (EGD) with biopsy revealed multiple polypoid masses in the body and antrum of the stomach, which histologically showed a dense chronic inflammatory infiltrate, consisting primarily of plasma cells. Subsequently the patient's reactive protein reagins (RPR) was found to be positive at a titer of 1:512. Silver stains of the gastric biopsy revealed numerous spirochetes in the lamina propria. Intravenous penicillin therapy was followed by prompt resolution of symptoms. This patient's clinical syndrome fulfills all of the criteria for the diagnosis of gastric syphilis. This case emphasizes that

the presentation and natural history of syphilis may be altered in patients with HIV-I coinfection. The implications regarding clinical management of patients with *Treponema pallidum* and HIV-I coinfection will be discussed.

Phlegmasia Cerulea Dolens: Familial Protein S Deficiency and Thrombolytic Therapy. Linda G. Philpot, MD, Donald Dinous, MD, Jesse Davison, MD, and Charles L. Crockett, Jr., MD, Roanoke.

Protein S is a vitamin K-dependent cofactor of activated protein C, which acts in the clotting cascade to inhibit clotting at the level of factors Va and VIIIa. Inheritable deficiency of protein S has been recognized since 1981, the clinical implication of which is thrombophilia. To our knowledge this is one of the first reported cases of phlegmasia cerulea dolens in the medical literature in a young patient with protein S deficiency.

A previously healthy 18-year-old athletic white male developed abrupt onset of a painful, blue left leg without obvious precipitating causes. Thigh diameters differed by 10 cm and the entire affected limb was grossly cyanotic, tender and edematous. Arterial pulses were present and confirmed by Doppler. Duplex scan demonstrated no flow in the left leg including iliac, common and deep femoral, popliteal and greater saphenous veins. Aggressive thrombolytic therapy with urokinase was initiated and improvement of symptoms and disappearance of cyanosis was noted within 12 hours. Slight flow in the deep femoral vein was present on the second hospital day and clinical improvement continued. After a 5-day course of thrombolytic therapy he was placed on IV heparin. Repeat Duplex scan demonstrated clot maturation and patency of the femoral system. He was discharged home anticoagulated on oral warfarin. Blood studies indicated absent free protein S and low normal levels of the bound (total) form. Further studies confirmed his father to have only a trace of protein S but no clinical events.

Young individuals presenting with venous thrombosis should be evaluated for inheritable and other hypercoagulable states. Thrombolytic therapy may be limb-saving in phlegmasia cerulea dolens.

Prevalence of and Variables Associated with Silent Ischemia on Exercise Thallium-201 Testing. Christine Gasperetti, MD, Lawrence Burwell, MD, and George Beller, MD, Charlottesville.

We prospectively studied 103 consecutive patients undergoing symptom-limited exercise thallium-201 testing to identify those variables which correlated with the presence of silent ischemia (SI). Fifty-nine patients (57%) had SI on testing. Silent ischemia was defined as presence of a TI-201 redistribution defect in absence of chest pain. There was no difference between SI and angina (AP) groups with respect to age,

sex, antianginal use, prevalence of diabetes mellitus, and exercise parameters including duration, METS or heart rate achieved, peak workload or peak double product.

A significantly higher percentage of SI patients had a recent myocardial infarction (31 vs 7%, p < 0.01), had no prior angina (64 vs 91%, p < 0.01), had dyspnea as an exercise endpoint (50 vs 35%, p < 0.05), and had redistribution in the supply regions of the right or left circumflex (50 vs 35%, p < 0.05) coronary arteries. Patients with AP had a significantly greater incidence of ST depression (64 vs 41%, p < 0.05). There was no difference between the two groups with respect to total TI-201 perfusion score, number of redistribution defects per patient, multivessel redistribution pattern, or extent of angiographic coronary artery disease (CAD).

In conclusion, we report a high prevalence of SI by exercise thallium testing. This occurs independently of exercise performance parameters, extent of angiographic CAD, and extent of exercise-induced scintigraphic defects.

Adenocarcinoma Presenting as Endocarditis. Brian Bachhuber, MD, and Munsey S. Wheby, MD, *Charlottesville*.

The hypercoagulable state and nonbacterial thrombotic endocarditis (NBTE) are known complications

of cancer. We present an unusual and instructive case of a previously healthy 44-year-old white woman with a clinical syndrome of fever, malaise, anemia, transient pleural effusions, splinter hemorrhages, hematuria/proteinuria, and acute onset of a left homonymous hemianopsia. Screening coagulation studies were normal. Head CT, abdominal CT, and abdominal aortogram were felt consistent with arterial embolic disease. Echocardiogram and Doppler study revealed an aortic vegetation with mild AI. Cultures of blood, urine and CSF were persistently negative and broad spectrum antibiotics failed to control the fever. Review of the abdominal CT suggested evidence of left deep vein thrombosis (DVT) and question of pulmonary infarction. Ultrasound showed bilateral leg DVT. Needle biopsy with ultrasound guidance of a hypodense liver lesion revealed poorly differentiated adenocarcinoma.

In this case of carcinoma-induced NBTE and hypercoagulability, the presentation was that of "culture negative endocarditis (CNE)." Initial management for the former, heparin, is contraindicated in bacterial endocarditis. We suggest that in CNE one must be wary of NBTE and the diseases associated with that condition—in this case, adenocarcinoma.

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VIRGINIA MEDICAL

Medical Care Here and There

ARE YOU dissatisfied with the way you must practice medicine? Do you hate government and big business controls? Have you had it up to here with insurance requirements? Are you constantly worried that some patient, dissatisfied with less than a perfect result, will institute a malpractice suit? If so, see the three-part "Medicine Abroad" in this issue; you are bound to feel better. For the physician, the United States remains the medical Valhalla.

What about the patient? Does the United States system supply optimal health care for all? Unfortunately, it does not, but optimal care is available. Our

problem lies in access to medical care. Both the British and Canadian health care systems provide excellent programs, but there is some question as to access in them; each appears to involve rationing of health care.

Sometime in March or early April 1990 (date and place not yet available), St. Mary's Hospital, Richmond, will sponsor a program including presentations of both the Canadian and British health care systems. The speakers will be authoritative and there will be a question and answer period. Watch for the announcement.

E.L.K. JR.

The Ideal Family Physician

I NEVER knew him, but over the years I have heard a lot of stories about him.

He was the ideal country doctor: capable, compassionate, sincere, always available, and very affordable. He had been well-trained at one of the larger medical schools, where he stayed for many years to teach. Eventually he could no longer tolerate the city and returned to his rural home in a nearby county.

By then he was an elderly bachelor with no living relatives and with few interests outside medicine. He always worked slowly and methodically, giving each and every patient a full share of his unhurried time.

His very modest home was next door to his even more modest one-room office which was so small that he had to go outside on the porch, winter or summer, while his women patients undressed or dressed.

He had a one-man operation with no overhead for nurse or secretary. He dispensed a few medications, but he did not need many because this was before our

pharmaceutical armamentarium had much therapeutic value. No insurance forms to fill out because there was no health insurance.

Even for those days his price was reasonable at \$.25 for children and \$.50 for adults, with liberal credit and plenty of charity. The hungry, down-on-their-luck people could always turn to him for a meal and a little pocket change.

He was greatly loved and respected in his community, and the people grieved when he became too ill and feeble to continue his medical practice, when he could no longer attend to their medical problems at any time of day or night, when they had to travel 20 miles to see someone who would charge them \$5 a visit. Oh, it was quite a shock to the community to see him close his office for the final time.

Only one year later he died, alone, in the county poor house for the elderly.

HENRY S. CAMPELL, MD

OBITUARY

Memoir of S. F. Driver 1905-1989

By Julian B. Doss, MD

Samuel Francis Driver, MD, was born September 20, 1905, in Staunton, Virginia, and died September 22, 1989, in Roanoke. He was a graduate of Bridgewater College, Class of 1927, where he was active in student government. He was business manager of the annual and captain of the football team. He taught at Elkton High School from 1927 to 1928 and was principal of Deerfield High School in 1928 and 1929. He matriculated at the Medical College of Virginia in the fall of 1929 and earned the MD degree in 1933. His initial practice at Troutville, Virginia, followed a brief internship at Lewis-Gale Hospital in 1933.

He was called to serve in the Army Medical Corps in January 1942 as captain and commanding officer of the 672nd Medical Collecting Company in the Italian Campaign April 1944 to August 1945 and was awarded the Bronze Star for meritorious service in the Po Valley breakthrough action.

On returning home, he established his practice on Williamson Road in 1946, where he served for the next 25 years. He was active in the progress of medicine. He served as secretary of the Blue Ridge Academy of Medicine and as secretary-treasurer of the Roanoke Academy of Medicine.

He was a member also of The Medical Society of Virginia, the Virginia Academy of Family Practice, and the American Medical Association.

Other activities included church Bible teacher, president of the Williamson Road Lions Club, member of the First Presbyterian Church, and other community affairs. He was an avid fisherman and played some golf. He joined the staff of the Veterans Administra-

tion Medical Center in Salem, Virginia, in 1960. He was acting chief of staff at the Mental Hygiene Clinic for the next 20 years. He received a commendation for meritorious service. On retiring, he continued part-time service with the Social Security Disability Board.

Dr. Driver was a beloved family physician, warm, caring and diligent. He was active in the advancement of medicine in days of limitations, unproven procedures and medications, and when travel was time-consuming and conditions difficult—at time impossible—in mountainous terrain and inclement weather.

He was blessed with a devoted, constant companion in his wife, Elizabeth Kinzie Drive, and celebrated their sixtieth anniversary a few days prior to his death. He is survived also by daughter, Eleanor Frances Driver Arnold, two grandsons, Charles Arnold III and Samuel Driver Arnold, and a sister, Rachael Driver Murphy.

• **Monford D. Custer, Jr., MD**, Winchester surgeon; Columbia University College of Physicians and Surgeons, 1940; age 74; died November 23, 1989.

• **Levi W. Hulley, Jr., MD**, Richmond; University of Virginia School of Medicine, 1939; age 75; died December 10, 1989. By specialty a family physician, Dr. Hulley had for many years been a medical administrator.

• **Edgar Willis Lacy, Jr., MD**, Winchester internist; Medical College of Virginia, 1936; age 81; died November 24, 1989.

• **Quentin J. Legg, MD**, Newport News radiologist; Medical College of Virginia, 1943; age 70; died November 16, 1989.

• **John J. O'Connor, MD**, retired anesthesiologist at Alexandria Hospital; State University of New York at Buffalo, 1950; age 69; died December 1, 1989. He was a past president of the Alexandria Medical Society.

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MEETINGS

February 23-25

Pediatric Emergency Medicine (Virginia Chapter, American Academy of Pediatrics and Virginia Pediatric Society), *Williamsburg*. 13 hrs. credit. Judy Suher, (804) 643-8130.

February 25-March 3

4th Annual Innovations in the Diagnosis and Treatment of Gastrointestinal Disorders (Georgetown University), *Vail, Colorado*. CME Office, 202-687-8735.

March 3

4th Annual Peripheral Vascular Disease Symposium (Fairfax Hospital/Georgetown University), *Falls Church*. 3 credit hrs. Fee, \$40. Cathy Sarraf, 703-698-2572.

March 5-8

Alton D. Brashear Postgraduate Course in Head and Neck Anatomy (Medical College of Virginia/VCU), *Richmond*. Hugo R. Seibel, PhD, 804-786-9624.

March 8-11

Controversies in Obstetrical Ultrasound (Eastern Virginia Medical School), *Snowbird, Utah*. 16 credit hrs. CME Office, 804-446-6140.

March 10-17

7th Annual Wilmer Institute Current Concepts in Ophthalmology (Johns Hopkins), *Vail, Colorado*. 30 credit hrs. Fee, \$475. CME Office, 301-955-2959.

March 11-14

Internal Medicine Seminar (Lloyd Noland Hospital), Disney World, *Lake Buena Vista, Florida*. CME Office, 205-783-5276.

March 13-14

13th Annual Symposium on Mental Health and the Law (University of Virginia Institute of Law, Psychiatry, Public Policy/Department Mental Health, Mental Retardation, Substance Abuse/Office of Virginia Attorney General), *Williamsburg*. Carolyn Englehard, 804-924-5435.

March 14-17

Pediatrics Seminar (Lloyd Noland Hospital), Disney World, *Lake Buena Vista, Florida*. CME Office, 205-783-5276.

March 15-17

Drugs, Behavior and the Brain: Advances in PET and SPECT Imaging (Johns Hopkins), *Baltimore*. 18 credit hrs. Fee, \$440. CME Office, 301-955-6046.

March 19-21

Spectrum of Developmental Disabilities XII: Prematurity—Neuro-Developmental Assessment and Intervention Issues (Kennedy Fellows Ass'n/Kennedy Institute Handicapped Children/Society Developmental Pediatrics/Johns Hopkins), *Baltimore*. 20 credit hrs. Juliet M. Nutt, 301-550-9423.

March 23

Sleep Apnea (Eastern Virginia Medical School), *Norfolk*. CME Office, 804-446-6140.

March 24-25

Advanced Cardiac Life Support Provider Course (Fairfax Hospital), *Falls Church*. Fee, \$200. Cathy Sarraf, 703-698-2572.

March 29-30

Diagnosis and Treatment of Neoplastic Disorders: Medical, Surgical and Radiotherapeutic Aspects (Johns Hopkins), *Baltimore*. 14.5 credit hrs. Fee, \$250. CME Office, 301-955-2959.

March 30-April 1

Pediatrics 1990 (American Academy of Pediatrics), *Marco Island, Florida*. Suzanne Goheen, 312-981-7884.

April 1-4

Conference on AIDS (Johns Hopkins), *Baltimore*. Registration, 1-800-334-8644.

April 4

37th Annual Clinical Conference (Obici Hospital), *Suffolk*. George J. Carroll, MD, 804-934-4000.

April 6-8

Mammography: Current Concepts and Interpretation (Eastern Virginia Medical School), *Williamsburg*. CME Office, 804-446-6140.

April 6-8

Annual Ophthalmology Conference: Cornea and External Disease (Medical College of Virginia/VCU), *Williamsburg*. CME Office, 804-786-0494.

April 19-21

Annual Meeting, Virginia Society of Otolaryngology/Head and Neck Surgery, *Alexandria*. Donna Scott, 804-353-2721.

April 19-22

10th Edition, Clinical Electrocardiography: Basic Concepts and Interpretation (Eastern Virginia Medical School), *New Orleans, Louisiana*. CME Office, 804-446-6140.

April 20-22

12th Annual Emergency Medicine for the Primary Care Physician (Medical College of Virginia/VCU), *Williamsburg*. CME Office, 804-786-0494.

April 21

Regular meeting of the Medical Society of Virginia's Council, *Richmond*. James L. Moore, Jr., 804-353-2721.

April 24

Common Problems in Primary Care (Fairfax Hospital), *Falls Church*. Cathy Sarraf, 703-698-2572.

April 26-29

11th Edition, Practical Dermatology for the Primary Care Physician (Eastern Virginia Medical School), *New Orleans, Louisiana*. CME Office, 804-446-6140.



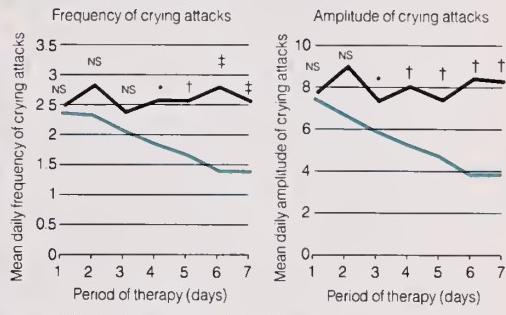
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YOCON® YOHIMBINE HCl

Description: Yohimbine is a 3a-15a-20B-17a-hydroxy Yohimbine-16a-carboxylic acid methyl ester. The alkaloid is found in Rubaceae and related trees. Also in Rauvolfia Serpentina (L) Benth. Yohimbine is an indolalkylamine alkaloid with chemical similarity to reserpine. It is a crystalline powder, odorless. Each compressed tablet contains (1/12 gr.) 5.4 mg of Yohimbine Hydrochloride.

Action: Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalamic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

Indications: Yocon® is indicated as a sympatholytic and mydriatic. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral α-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.^{1,2} Also dizziness, headache, skin flushing reported when used orally.^{1,3}

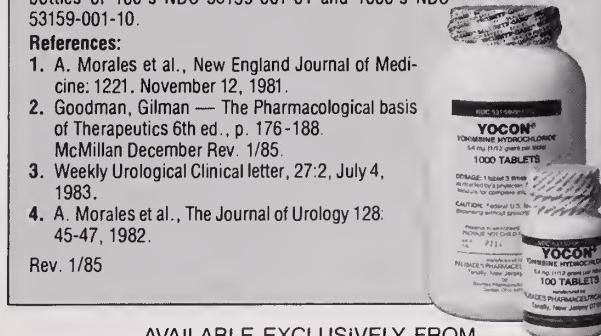
Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.^{1,3,4} 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to 1/2 tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.³

How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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VASOTEC® (ENALAPRIL MALEATE | MSD)

VASOTEC is available in 2.5-mg, 5-mg, 10-mg, and 20-mg tablet strengths.

Contraindications: VASOTEC® (Enalapril Maleate, MSD) is contraindicated in patients who are hypersensitive to this product and in patients with a history of angioedema related to previous treatment with an ACE inhibitor.

Warnings: **Angioedema:** Angioedema of the face, extremities, lips, tongue, glottis, and/or larynx has been reported in patients treated with ACE inhibitors, including VASOTEC. In such cases, VASOTEC should be promptly discontinued and the patient carefully observed until the swelling disappears. In instances where swelling has been confined to the face and lips, the condition has generally resolved without treatment, although antihistamines have been useful in relieving symptoms. Angioedema associated with laryngeal edema may be fatal. **Where there is involvement of the tongue, glottis, or larynx likely to cause airway obstruction, appropriate therapy, e.g., subcutaneous epinephrine solution 1:1000 (0.3 mL to 0.5 mL), should be promptly administered.** (See ADVERSE REACTIONS.)

Hypotension: Excessive hypotension is rare in uncomplicated hypertensive patients treated with VASOTEC alone. Patients with heart failure given VASOTEC commonly have some reduction in blood pressure, especially with the first dose, but discontinuation of therapy for continuing symptomatic hypotension usually is not necessary when dosing instructions are followed. Caution should be observed when initiating therapy. (See DOSAGE AND ADMINISTRATION.) Patients at risk for excessive hypotension, sometimes associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death, include those with the following conditions or characteristics: heart failure, hyponatremia, high-dose diuretic therapy, recent intensive diuresis or increase in diuretic dose, renal dialysis, or severe volume and/or salt depletion on any etiology. It may be advisable to eliminate the diuretic (except in patients with heart failure), reduce the diuretic dose, or increase salt intake cautiously before initiating therapy with VASOTEC in patients at risk for excessive hypotension who are able to tolerate such adjustments. (See PRECAUTIONS, Drug Interactions and ADVERSE REACTIONS.) In patients at risk for excessive hypotension, therapy should be started under very close medical supervision and such patients should be followed closely for the first two weeks of treatment and whenever the dose of enalapril and/or diuretic is increased. Similar considerations may apply to patients with ischemic heart disease or cardiovascular disease in whom an excessive fall in blood pressure could result in a myocardial infarction or cerebrovascular accident. If excessive hypotension occurs, the patient should be placed in the supine position and, if necessary, receive an intravenous infusion of normal saline. A transient hypotensive response is not a contraindication to further doses of VASOTEC, which usually can be given without difficulty once the blood pressure has stabilized. If symptomatic hypotension develops, a dose reduction or discontinuation of VASOTEC or concomitant diuretic may be necessary.

Neutropenia/Agranulocytosis: Another ACE inhibitor, captopril, has been shown to cause agranulocytosis and bone marrow depression, rarely in uncomplicated patients but more frequently in patients with renal impairment, especially if they also have a collagen vascular disease. Available data from clinical trials of enalapril are insufficient to show that enalapril does not cause agranulocytosis at similar rates. Foreign marketing experience has revealed several cases of neutropenia or agranulocytosis in which a causal relationship to enalapril cannot be excluded. Periodic monitoring of white blood cell counts in patients with collagen vascular disease and renal disease should be considered.

Precautions: General Impaired Renal Function: As a consequence of inhibiting the renin-angiotensin-aldosterone system, changes in renal function may be anticipated in susceptible individuals. In patients with severe heart failure whose renal function may depend on the activity of the renin-angiotensin-aldosterone system, treatment with ACE inhibitors, including VASOTEC, may be associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death.

In clinical studies in hypertensive patients with unilateral or bilateral renal artery stenosis, increases in blood urea nitrogen and serum creatinine were observed in 20% of patients. These increases were almost always reversible upon discontinuation of enalapril and/or diuretic therapy. In such patients, renal function should be monitored during the first few weeks of therapy.

Some patients with hypertension or heart failure with no apparent preexisting renal vascular disease have developed increases in blood urea and serum creatinine, usually minor and transient, especially when VASOTEC has been given concomitantly with a diuretic. This is more likely to occur in patients with preexisting renal impairment. Dosage reduction and/or discontinuation of the diuretic and/or VASOTEC may be required.

Evaluation of patients with hypertension or heart failure should always include assessment of renal function. (See DOSAGE AND ADMINISTRATION.)

Hyperkalemia: Elevated serum potassium (>5.7 mEq/L) was observed in approximately 1% of hypertensive patients in clinical trials. In most cases, these were isolated values which resolved despite continued therapy. Hyperkalemia was a cause of discontinuation of therapy in 0.28% of hypertensive patients. In clinical trials in heart failure, hyperkalemia was observed in 3.8% of patients, but was not a cause for discontinuation.

Risk factors for the development of hyperkalemia include renal insufficiency, diabetes mellitus, and the concomitant use of potassium-sparing diuretics, potassium supplements, and/or potassium-containing salt substitutes, which should be used cautiously, if at all, with VASOTEC. (See Drug Interactions.)

Surgery/Anesthesia: In patients undergoing major surgery or during anesthesia with agents that produce hypotension, enalapril may block angiotensin II formation secondary to compensatory renin release. If hypotension occurs and is considered to be due to this mechanism, it can be corrected by volume expansion.

Information for Patients

Angioedema: Angioedema, including laryngeal edema, may occur especially following the first dose of enalapril. Patients should be so advised and told to report immediately any signs or symptoms suggesting angioedema (swelling of face, extremities, eyes, lips, tongue, difficulty in swallowing or breathing) and to take no more drug until they have consulted with the prescribing physician.

Hypotension: Patients should be cautioned to report lightheadedness, especially during the first few days of therapy. If actual syncope occurs, the patients should be told to discontinue the drug until they have consulted with the prescribing physician.

All patients should be cautioned that excessive perspiration and dehydration may lead to an excessive fall in blood pressure because of reduction in fluid volume. Other causes of volume depletion such as vomiting or diarrhea may also lead to a fall in blood pressure; patients should be advised to consult with the physician.

Hyperkalemia: Patients should be told not to use salt substitutes containing potassium without consulting their physician.

Neutropenia: Patients should be told to report promptly any indication of infection (e.g., sore throat, fever) which may be a sign of neutropenia.

NOTE: As with many other drugs, certain advice to patients being treated with enalapril is warranted. This information is intended to aid in the safe and effective use of this medication. It is not a disclosure of all possible adverse or intended effects.

Drug Interactions

Hypotension: Patients on diuretics and especially those in whom diuretic therapy was recently instituted may occasionally experience an excessive reduction of blood pressure after initiation of therapy with enalapril. The possibility of hypotensive effects with enalapril can be minimized by either discontinuing the diuretic or increasing the salt intake prior to initiation of treatment with enalapril. If it is necessary to continue the diuretic, provide close medical supervision after the initial dose for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and DOSAGE AND ADMINISTRATION.)

Agents Causing Renin Release: The antihypertensive effect of VASOTEC is augmented by antihypertensive agents that cause renin release (e.g., diuretics).

Other Cardiovascular Agents: VASOTEC has been used concomitantly with beta-adrenergic-blocking agents, methyldopa, nitrates, calcium-blocking agents, hydralazine, prazosin, and digoxin without evidence of clinically significant adverse interactions.

Agents Increasing Serum Potassium: VASOTEC attenuates potassium loss caused by thiazide-type diuretics. Potassium-sparing diuretics (e.g., spironolactone, triamterene, or amiloride), potassium supplements, or potassium-containing salt substitutes may lead to significant increases in serum potassium. Therefore, if concomitant use of these agents is indicated because of demonstrated hypokalemia, they should be used with caution and with frequent monitoring of serum potassium. Potassium-sparing agents should generally not be used in patients with heart failure receiving VASOTEC.

Lithium: Lithium toxicity has been reported in patients receiving lithium concomitantly with drugs which cause elimination of sodium, including ACE inhibitors. A few cases of lithium toxicity have been reported in patients receiving concomitant VASOTEC and lithium and were reversible upon discontinuation of both drugs. It is recommended that serum lithium levels be monitored frequently if enalapril is administered concomitantly with lithium.

Pregnancy - Category C: There was no fetotoxicity or teratogenicity in rats treated with up to 200 mg/kg/day of enalapril (333 times the maximum human dose). Fetal toxicity, expressed as a decrease in average fetal weight, occurred in rats given 1200 mg/kg/day of enalapril but did not occur when these animals were supplemented with saline. Enalapril was not teratogenic in rabbits. However, maternal and fetal toxicity occurred in some rabbits at doses of 1 mg/kg/day or more. Saline supplementation prevented the maternal and fetal toxicity seen at doses of 3 and 10 mg/kg/day, but not at 30 mg/kg/day (50 times the maximum human dose).

Radioactivity was found to cross the placenta following administration of labeled enalapril to pregnant hamsters. There are no adequate and well-controlled studies of enalapril in pregnant women. However, data are available that

show enalapril crosses the human placenta. Because the risk of fetal toxicity with the use of ACE inhibitors has not been clearly defined, VASOTEC® (Enalapril Maleate, MSD) should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Postmarketing experience with all ACE inhibitors thus far suggests the following with regard to pregnancy outcome. Inadvertent exposure limited to the first trimester of pregnancy has not been reported to affect fetal outcome adversely. Fetal exposure during the second and third trimesters of pregnancy has been associated with fetal and neonatal morbidity and mortality.

When ACE inhibitors are used during the later stages of pregnancy, there have been reports of hypotension and decreased renal perfusion in the newborn. Oligohydramnios in the mother has also been reported, presumably representing decreased renal function in the fetus. Infants exposed *in utero* to ACE inhibitors should be closely observed for hypotension, oliguria, and hypokalemia. If oliguria occurs, attention should be directed toward support of blood pressure and renal perfusion with the administration of fluids and pressors as appropriate. Problems associated with prematurity such as patent ductus arteriosus have occurred in association with maternal use of ACE inhibitors, but it is not clear whether they are related to ACE inhibition, maternal hypertension, or the underlying prematurity.

Nursing Mothers: Milk in lactating rats contains radioactivity following administration of ¹⁴C-enalapril maleate. It is not known whether this drug is secreted in human milk. Because many drugs are secreted in human milk, caution should be exercised when VASOTEC is given to a nursing mother.

Pediatric Use: Safety and effectiveness in children have not been established.

Adverse Reactions: VASOTEC has been evaluated for safety in more than 10,000 patients, including over 1000 patients treated for one year or more. VASOTEC has been found to be generally well tolerated in controlled clinical trials involving 2987 patients.

HYPERTENSION: The most frequent clinical adverse experiences in controlled trials were headache (5.2%), dizziness (4.3%), and fatigue (3%).

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in controlled clinical trials were diarrhea (1.4%), nausea (1.4%), rash (1.4%), cough (1.3%), orthostatic effects (1.2%), and asthenia (1%).

HEART FAILURE: The most frequent clinical adverse experiences in both controlled and uncontrolled trials were dizziness (7.9%), hypotension (6.7%), orthostatic effects (2.2%), syncope (2.2%), cough (2.2%), chest pain (2.1%), and diarrhea (2.1%).

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in both controlled and uncontrolled clinical trials were fatigue (7.8%), headache (1.8%), abdominal pain (1.6%), asthenia (1.5%), orthostatic hypotension (1.6%), vertigo (1.6%), angina pectoris (1.5%), nausea (1.3%), vomiting (1.3%), bronchitis (1.3%), dyspnea (1.3%), urinary tract infection (1.3%), rash (1.3%), and myocardial infarction (1.2%).

Other serious clinical adverse experiences occurring since the drug was marketed or adverse experiences occurring in 0.5% to 1% of patients with hypertension or heart failure in clinical trials in order of decreasing severity within each category.

Cardiovascular: Cardiac arrest, myocardial infarction or cerebrovascular accident, possibly secondary to excessive hypotension in high-risk patients (see WARNINGS, Hypotension), cardiac arrest, pulmonary embolism and intarction, rhythm disturbances, atrial fibrillation, palpitation.

Digestive: Ileus, pancreatitis, hepatitis or cholestatic jaundice, melena, anorexia, dyspepsia, constipation, glossitis, stomatitis.

Musculoskeletal: Muscle cramps.

Nervous/Psychiatric: Depression, confusion, ataxia, somnolence, insomnia, nervousness, paresthesia.

Urogenital: Renal failure, oliguria, renal dysfunction (see PRECAUTIONS and DDSAGE AND ADMINISTRATION).

Respiratory: Bronchospasm, rhinorrhea, sore throat and hoarseness, asthma, upper respiratory infection.

Skin: Herpes zoster, urticaria, pruritus, alopecia, flushing, hyperhidrosis.

Special Senses: Blurred vision, taste alteration, tinnitus.

A symptom complex has been reported which may include a positive ANA, an elevated erythrocyte sedimentation rate, arthralgia/arthritis, myalgias, fever, serositis, vasculitis, leukocytosis, eosinophilia, photosensitivity, rash and other dermatologic manifestations.

Angioedema: Angioedema has been reported in patients receiving VASOTEC (0.2%). Angioedema associated with laryngeal edema may be fatal. Angioedema of the face, extremities, lips, tongue, glottis, and/or larynx occurs, treatment with VASOTEC should be discontinued and appropriate therapy instituted immediately. (See WARNINGS.)

Hypotension: In the hypertensive patients, hypotension occurred in 0.9% and syncope occurred in 0.5% of patients following the initial dose or during extended therapy. Hypotension or syncope was a cause for discontinuation of therapy in 0.1% of hypertensive patients. In heart failure patients, hypotension occurred in 6.7% and syncope occurred in 2.2% of patients. Hypotension or syncope was a cause for discontinuation of therapy in 1.9% of patients with heart failure. (See WARNINGS.)

Clinical Laboratory Test Findings

Serum Electrolytes: Hyperkalemia (see PRECAUTIONS), hyponatremia.

Creatinine, Blood Urea Nitrogen: In controlled clinical trials, minor increases in blood urea nitrogen and serum creatinine, reversible upon discontinuation of therapy, were observed in about 0.2% of patients with essential hypertension treated with VASOTEC alone. Increases are more likely to occur in patients receiving concomitant diuretics or in patients with renal artery stenosis. (See PRECAUTIONS.) In patients with heart failure who were also receiving diuretics with or without digitalis, increases in blood urea nitrogen or serum creatinine, usually reversible upon discontinuation of VASOTEC and/or other concomitant diuretic therapy, were observed in about 11% of patients. Increases in blood urea nitrogen or creatinine were a cause for discontinuation in 1.2% of patients.

Hemoglobin and Hematocrit: Small decreases in hemoglobin and hematocrit (mean decreases of approximately 0.3 g/dL and 1.0 vol.-%, respectively) occur frequently in either hypertension or heart failure patients treated with VASOTEC but are rarely of clinical importance unless another cause of anemia coexists. In clinical trials less than 0.1% of patients discontinued therapy due to anemia.

Other (Causal Relationship Unknown): In marketing experience, rare cases of neutropenia, thrombocytopenia, and bone marrow depression have been reported. A few cases of hemolysis have been reported in patients with G6PD deficiency.

Liver Function Tests: Elevations of liver enzymes and/or serum bilirubin have occurred.

Dosage and Administration: Hypertension: In patients who are currently being treated with a diuretic, symptomatic hypotension occasionally may occur following the initial dose of VASOTEC. The diuretic should, if possible, be discontinued for two or three days before beginning therapy with VASOTEC to reduce the likelihood of hypotension. (See WARNINGS.) If the patient's blood pressure is not controlled with VASOTEC alone, diuretic therapy may be resumed.

If the diuretic cannot be discontinued, an initial dose of 2.5 mg should be used under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and PRECAUTIONS.)

The recommended initial dose in patients not on diuretics is 5 mg once a day. Dosage should be adjusted according to blood pressure response. The usual dosage range is 10 to 40 mg per day administered in a single dose or in two divided doses. In some patients treated once daily, the antihypertensive effect may diminish toward the end of the dosing interval. In such patients, an increase in dosage or twice-daily administration should be considered. If blood pressure is not controlled with VASOTEC alone, a diuretic may be added.

Concomitant administration of VASOTEC with potassium supplements, potassium salt substitutes, or potassium-sparing diuretics may lead to increases of serum potassium (see PRECAUTIONS).

Dosage Adjustment in Hypertensive Patient with Renal Impairment: The usual dose of enalapril is recommended for patients with a creatinine clearance > 30 mL/min (serum creatinine of up to approximately 3 mg/dL). For patients with creatinine clearance ≤ 30 mL/min (serum creatinine ≥ 3 mg/dL), the first dose is 2.5 mg once daily. The dosage may be titrated upward until blood pressure is controlled or to a maximum of 40 mg daily.

Heart Failure: VASOTEC is indicated as adjunctive therapy with diuretics and digitalis. The recommended starting dose is 2.5 mg once or twice daily. After the initial dose of VASOTEC, the patient should be observed under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and PRECAUTIONS, Drug Interactions.) If possible, the dose of the diuretic should be reduced, which may diminish the likelihood of hypotension. The appearance of hypotension after the initial dose of VASOTEC does not preclude subsequent careful dose titration with the drug, following effective management of the hypotension. The usual therapeutic dose range for the treatment of heart failure is 5 to 20 mg daily given in two divided doses. The maximum daily dose is 40 mg. Once-daily dosing has been effective in a controlled study, but nearly all patients in this study were given 40 mg, the maximum recommended daily dose, and there has been much more experience with twice-daily dosing. In addition, in a placebo-controlled study which demonstrated reduced mortality in patients with severe heart failure (NYHA Class IV), patients were treated with 2.5 to 40 mg per day of VASOTEC, almost always administered in two divided doses. (See CLINICAL PHARMACOLOGY, Pharmacodynamics and Clinical Effects.) Dosage may be adjusted depending upon clinical or hemodynamic response. (See WARNINGS.)

Dosage Adjustment in Patients with Heart Failure and Renal Impairment or Hyponatremia: In patients with heart failure who have hyponatremia (serum sodium < 130 mEq/L) or with serum creatinine ≥ 1.6 mg/dL, therapy should be initiated at 2.5 mg daily under close medical supervision. (See DOSAGE AND ADMINISTRATION, Heart Failure, WARNINGS, and PRECAUTIONS, Drug Interactions.) The dose may be increased to 2.5 mg b.i.d., then 5 mg b.i.d. and higher as needed, usually at intervals of four days or more. If at the time of dosage adjustment there is not excessive hypotension or significant deterioration of renal function, the maximum daily dose is 40 mg.

For more detailed information, consult your MSD Representative or see Prescribing Information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, PA 19486

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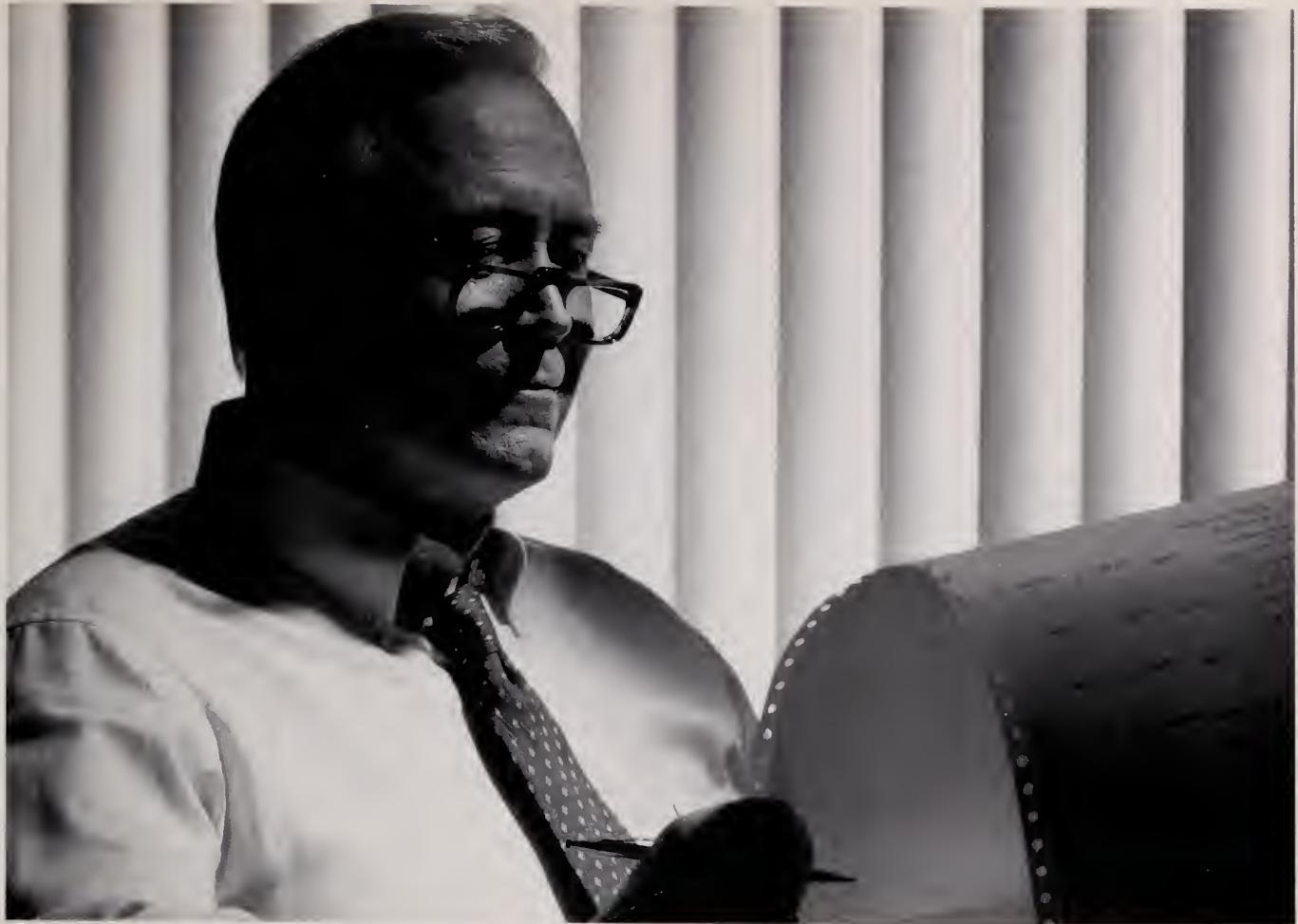
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MEDIBYTES

NEWS FROM VA MED'S COMPUTER ROOM

COUNCIL'S NEW YEAR OFF TO GOOD START

A string of success stories brightened the agenda of the Medical Society of Virginia Council's first meeting of 1990 on January 20 in Richmond. The new MSV President, **Dr. William H. Barney**, presided.

Brightest and best of all was the news of the upturn in membership. Deunification triggered the turnaround, reported **Dr. Jeffrey W. Wilson**, chairman, Membership Committee, citing a big bunch of reinstatement requests and a surge of new members. All are listed by name on pages 94-101 of this issue, and Dr. Wilson forecast more to come when applications still outstanding are heard from...From the MSVRO via **Terry Dwyer**, exec director, came word of a small but significant success: Medicare precertification of certain surgical procedures has been modified to allow two working days after an emergency operation in which to obtain approval. The Council had worked on this sticky wicket, and so had the MSV and AMA Houses...All the signs of success are being exhibited by the Teen Health Forum in work under the auspices of the Auxiliary, **Mrs. Vivian Petty**, MSVA President, assured the councilors. Over 77 high schools over the state have expressed interest in participating in the event on March 20 in Richmond...In the solid success column, enter VaMPAC's reception for Virginia's legislators on the eve of the General Assembly's opening, said **Dr. Read F. McGehee, Jr.**, chairman elect, where a good time was had by a big crowd of legislators and physicians. Also solidly successful: VaMPAC's membership department, with a jump from 500 to 600 members. Another deunification bonus?...Two recent Medical Society of Virginia surveys/reports are rousing success stories, with lots of media coverage, all of it good, and respectful receptions by public health officials and the state legislature, counsel **Sandra Kramer** told the councilors. The one titled, "Problems and Solutions to Access to Obstetrical Care in Virginia" supported the medical community's perception that the problems of ob access are a result of malpractice costs, potential lawsuits, and the low rates and administrative hassles associated with reimbursement. Access to primary care was the subject of the second survey/report; by unanimous vote the councilors established an official



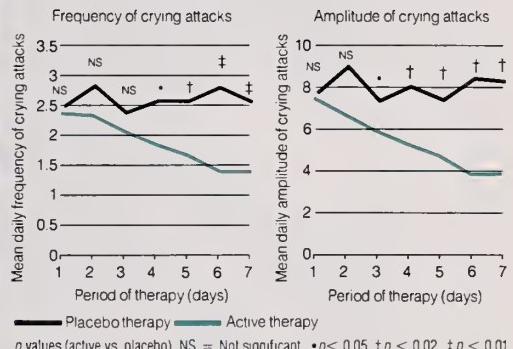
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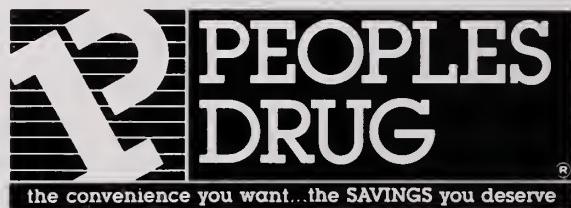
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Present at Council's first meeting of the New Year were these new members: from left, Dr. Louis D. Parham, councilor, and Dr. Norman R. Edwards, vice councilor, 1st district; Dr. James A. Shield, Jr., councilor, and Dr. Lawrence E. Blanchard, III, vice councilor, 3rd district.

MSV position on the subject by endorsing that report's findings. Also in the area of primary care, the councilors adopted fourteen recommendations and a resolution from the Rural Health Committee, **Dr. Charles H. Crowder, Jr.**, chairman. They had to do with such problems as the supply of primary care physicians, inequities in reimbursement in rural areas, including low Medicaid fees, and protecting PCPs in solo practice from insurance prejudice and litigation problems...The Public Relations Committee, **Dr. William L. Harp**, chairman, was successful in its quest for an appropriation of \$2,000 for the production of a public service announcement to be distributed statewide. On recommendation of the Executive Committee, Council okayed the request...To beef up the Society's membership recruitment and lobbying efforts, Dr. Barney announced, the Executive Committee wanted to solicit the part-time services of **Dr. James F. Ghaphery**, retired anesthesiologist and veteran contributor to countless MSV lobbying efforts. Was Council willing? You bet, responded the councilors. The solicitation was a success, and Dr. Ghaphery is now on staff as the Society's Field Representative. --A.G.



NEW AND REINSTATED MSV MEMBERS

A healthy upturn in Medical Society of Virginia membership was the instant response to MSV President William H. Barney's letter of November 21 announcing the end of four years of MSV/AMA unification. Receipts of applications of new memberships jumped from 58 for October-November to 78 for December-January, and as of February 1 a total of 262 lapsed members had requested reinstatement. The new members appear here as we usually publish them, according to component society of origin. The reinstated members are listed in the order in which their requests to rejoin were received.

Albemarle County Medical Society

David L. Bogdonoff, MD, General Surgery, 2100 Devonshire Road, Charlottesville VA 22901

R. J. Canterbury II, MD, Psychiatry/Neurology, Blue Ridge Hospital, 6 East Drawer D, Charlottesville VA 22901

Charles W. Gross, MD, Otolaryngology, Box 430, University of Virginia Health Sciences Center, Charlottesville VA 22908

Stuart S. Howards, MD, Urology, Box 422, University of Virginia Health Sciences Center, Charlottesville VA 22908

William F. Tompkins III, MD, Gastroenterology, 1100 East Jefferson Street, Charlottesville VA 22901

Alexandria Medical Society

Mark R. Fracasso, MD, Obstetrics/Gynecology, 4801 Kenmore Avenue, Alexandria VA 22304

Alleghany-Bath County Medical Society

James E. Ballou, MD, Internal Medicine, PO Drawer 547, Clifton Forge VA 24422

Mary Jane Luke, MD, Pediatric Cardiology, PO Box 351, Covington VA 24426

Arlington County Medical Society

Todd P. Berner, MD, Obstetrics/Gynecology, 2501 North Glebe Road, Arlington VA 22202

James C. McFalls, MD, Obstetrics/Gynecology, 1715 North George Mason Drive, Arlington VA 22205

Chesapeake Medical Society

Erik H. Kisa, MD, Emergency Medicine, 219-B 55th Street, Virginia Beach VA 23451

David A. Pitrolo, MD, Emergency Medicine, 524 Wickwood Drive, Chesapeake VA 23320

Lawrence E. Valentine, MD, Emergency Medicine, 7 Schooner Ridge, Kitty Hawk NC 27949

Bruce D. Waldholtz, MD, Gastroenterology, 300 Medical Parkway, Chesapeake VA 23320

Francis E. Watson, MD, Emergency Medicine, 513 Woodards Ford Road, Chesapeake VA 23320

Thomas V. Whelan, MD, Nephrology, 3033 Princess Anne Crescent, Chesapeake VA 23321

Danville-Pittsylvania Academy of Medicine

Bhushan H. Pandya, MD, Gastroenterology, 501 Rison Street, Danville VA 24541

Y. H. Charles Suh, MD, Cardiology, 115 South Main Street, Danville VA 24541

Fairfax County Medical Society

Robert A. Albus, MD, Cardiovascular/Thoracic Surgery, 3301 Woodburn Road, Annadale VA 22003

Allyson A. Askew, MD, Pediatric Surgery, 3301 Woodburn Road, Annandale VA 22003

John J. Basile, MD, Urology, 3299 Woodburn Road, Annandale VA 22003

Edward E. Eder, MD, Internal Medicine, 1015 Emerald Drive, Fairfax VA 22308

Ian H. Leibowitz, MD, Pediatric Gastroenterology, 3027 Javier Road, Fairfax VA 22031



Eveline B. Marguardt, MD, Obstetrics/Gynecology, 1800 Town Center Drive, Reston VA 22090
Mark D. Vickers, MD, Internal Medicine, 112 Elden Street, Herndon VA 22070

Fredericksburg Area Medical Society
Frank J. Durcan, MD, Obstetrics/Gynecology, 1701 Fall Hill Avenue, Fredericksburg VA 22401

Hampton Medical Society
Paul W. Popish, MD, Pediatrics, 304 Marcella Road, Hampton VA 23666

Loudoun County Medical Society
Salvatore N. Amari, MD, Internal Medicine, 211 Gibson Street NW, Leesburg VA 22075
Russell H. Mitchell, MD, Dermatology, 823-J South King Street, Leesburg VA 22075

Lynchburg Academy of Medicine
David W. Frantz, MD, Thoracic Surgery, 2000 Tate Springs Road, Lynchburg VA 24501
Morris E. McCrary III, MD, Neurosurgery, 1922 Thomson Drive, Lynchburg VA 24501
Gerald S. Modjeska, MD, Occupational Medicine, 200 Howard Drive, Lynchburg VA 24503
William H. Van Dyke Jr., MD, Cardiology, 2215 Landover Place, Lynchburg VA 24501

Mid-Tidewater Medical Society
James R. Robusto, MD, Family Practice, PO Box 880, Urbanna VA 23175

Newport News Medical Society
Heather A. Argyle, MD, Gynecology, 606 Denbigh Boulevard, Newport News VA 23602
Laurel G. Weaver, MD, Anesthesiology, 60 Chowning Drive, Hampton VA 23664

Norfolk Academy of Medicine
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Curtis A. Winter, MD, Obstetrics/Gynecology, 1330 Amherst Street, Winchester VA 22601

Patrick-Henry Medical Society
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Portsmouth Academy of Medicine
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Adrienne L. Coqueran, MD, Internal/Emergency Medicine, 921 Hanover Drive, Virginia Beach VA 23464
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Marc R. Katz, MD, Cardiac Surgery, 7107 Jahnke Road, Richmond VA 23225
Michael K. Kyles, MD, Orthopedic Surgery, 700 West Grace Street, Richmond VA 23220
David W. Richardson, MD, Cardiology, Box 128, MCV Station, Richmond VA 23298

Rockingham County Medical Society
Glenn E. Deputy, MD, Neurology, 170 South Main Street, Harrisonburg VA 22801
Mark A. Kniss, MD, Family Practice, 1000 Chicago Avenue, Harrisonburg VA 22801
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Southwestern Virginia Medical Society
Larry A. Cowley, MD, Otolaryngology, 508 South Main Street, Blacksburg VA 24060

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Gregory J. Endres-Bercher, MD, Internal/Emergency Medicine, 215 Tower Street, Tazewell VA 24651
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Preston L. Judson, MD, Cardiology, 707 Gittings Street, Suffolk VA 23434
Stephen S. Marsh, MD, Family Practice, Smithfield Medical Clinic, Smithfield VA 23430

Virginia Beach Medical Society

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Martin G. O'Grady, MD, General/Vascular Surgery, PO Box 4159, Virginia Beach VA 23454

Williamsburg-James City County Medical Society

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Richard G. Samaha, MD, Emergency Medicine, 111 Clarendon Court, Williamsburg VA 23185
David G. Teasley, MD, Plastic Surgery, 1315 Jamestown Road, Williamsburg VA 23185

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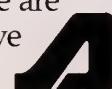
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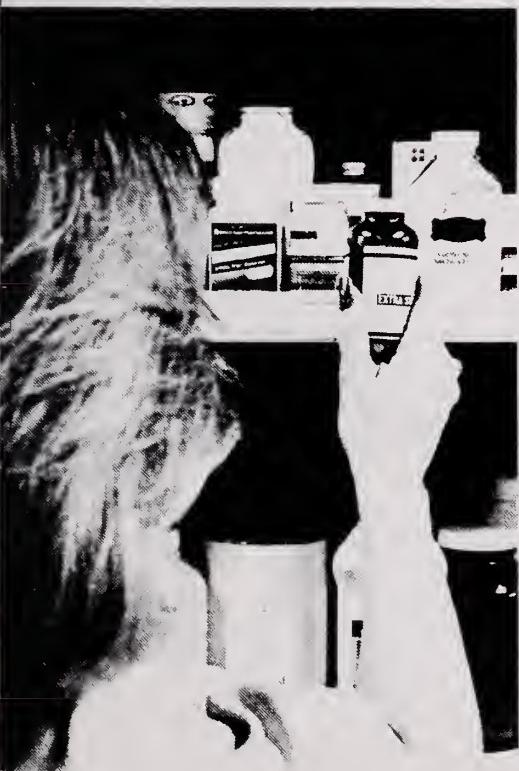
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Coronary Artery Bypass Surgery: Emerging Trends in Mortality

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In a consecutive series of 4,697 patients undergoing coronary artery bypass surgery, these risk factors were found to be significant for increased postoperative mortality: age greater than 70, female sex, unstable angina, prior myocardial infarction, hypertension, diabetes mellitus, and ejection fraction less than .40. A comparison by year (1980-1988) revealed a steadily increasing incidence of these risk factors. Future analyses of coronary artery bypass mortality should include risk-factor stratification.

FOllowing the introduction of coronary artery bypass surgery in 1968,¹ reports in the 1970s and early 1980s continued to document the decreasing risks of coronary artery bypass surgery.² Indeed, some institutions involved in the collaborative study in coronary artery surgery (CASS) reported overall operative mortality rates on patients operated between 1974 and 1979 of less than 1%.² More recently, however, many anecdotal and some documented reports have addressed a trend toward surgical revascularization for high-risk patients and medical management or transluminal angioplasty for low-risk patients, resulting in higher recent operative mortality rates.³ To analyze the effects of this trend on current surgical mortality at our institution, we examined the mortality of coronary artery bypass surgery in the 1980s in relation to known or suspected risk factors.

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Methods

Clinical and demographic data on all patients undergoing cardiac surgery at Fairfax Hospital, Falls Church, Virginia, are entered into a computerized data base using File Manager software and a DEC 11/24 computer. From January 1, 1980, through December 31, 1988, a total of 4,697 patients who had undergone primary, isolated coronary artery bypass surgery, including emergencies, were so entered. By definition this excluded patients undergoing redo surgery or patients having combined coronary artery bypass and cardiac valve procedures. Postoperative mortality, defined as inhospital mortality regardless of postoperative day, was recorded and analyzed in relation to suspected preoperative risk factors (Table 1) using standard chi-square analysis to compare the mortality of all patients in the series with a given risk factor to those without that risk factor. Following the identification of significant preoperative risk factors for increased surgical mortality, the incidence of these risk factors were analyzed by year.

Results

There were 3,482 males and 855 females whose age

and sex by year are noted in Figures 1 and 2. Overall operative mortality rate was 2.5%. Significant predictors of increased postoperative mortality are listed in Table 2. Not found to be significant were prior angioplasty, a history of cigarette smoking, or a family history of coronary artery disease.

The incidence by year of patients presenting with unstable or postmyocardial infarction angina, diabetes, and hypertension are shown in Figure 3A, and the incidence by year of female sex, ejection fraction less

than .4, age greater than 70, and prior myocardial infarction are shown in Figure 3B. Particularly striking is the increasing incidence of hypertension, unstable angina, age greater than 70 and ejection fraction less than .4 in this surgical population. The trend toward increasing operative mortality is shown in Figure 4.

Discussion

A number of studies have documented the increased risk of coronary artery bypass surgery in the elderly, the clinically unstable, and those with impaired left ventricular function.⁴⁻⁹ Females have a well-documented increase in risk, although this may be related to size more than sex.^{10,11} Diabetes in some series has been associated with more extensive disease and increased operative risks independent of other factors.¹² Prior myocardial infarction was not assessed independently from left ventricular function and it is conceivable that prior myocardial infarction is a risk factor only as it relates to reduced ejection fraction. Hypertension is more common in the elderly and diabetics and its validity as an independent risk factor cannot be supported in this study.¹²

The increasing incidence of "high-risk" patients undergoing coronary artery bypass surgery found in this study is probably due to a number of factors, including the general aging of the population and the diminishing reluctance on the part of referring cardiologists to refer older, sicker patients for surgery. The fact that the "sickest" patients, e.g., those with severely diminished left ventricular function, may indeed represent a subgroup that has the most to gain from coronary artery bypass surgery is becoming well accepted.¹³ Finally, the role of coronary angioplasty cannot be overemphasized. Davis and coworkers recently compared the mortality of coronary artery bypass surgery before and after the advent of angioplasty. They discovered the latter group to be significantly older with more prior myocardial infarctions, more co-morbidity, and a four-fold incidence of emergency surgery with an increased operative mortality rate.¹⁴

Table 1. Risk Factors Analyzed.

Prior angioplasty	Smoking history
Diabetes mellitus	Family history coronary artery disease
Unstable angina*	Age >70
Prior myocardial infarction	Ejection fraction <.4
Hypertension	Sex

* Defined as persistent or recurrent angina at rest despite maximal medical therapy. The vast majority of these patients are on intravenous nitrates preoperatively.

Table 2. Significant Risk Factors for Increased Operative Mortality

Variable	Overall Mortality (%)	p value
Diabetes mellitus	5.3	<.001
No diabetes mellitus	2.2	
Unstable angina	5.6	<.001
Not unstable	1.9	
Prior myocardial infarction	3.6	<.001
No prior myocardial infarction	1.7	
Hypertension	3.4	<.05
No hypertension	2.1	
Age >70	8.4	<.001
Age ≤70	1.9	
Ejection fraction .4	4.0	<.001
Ejection fraction <.4	2.1	
Female sex	6.0	<.001
Male sex	1.9	

Fig. 1. Patient Population by Year and Sex.

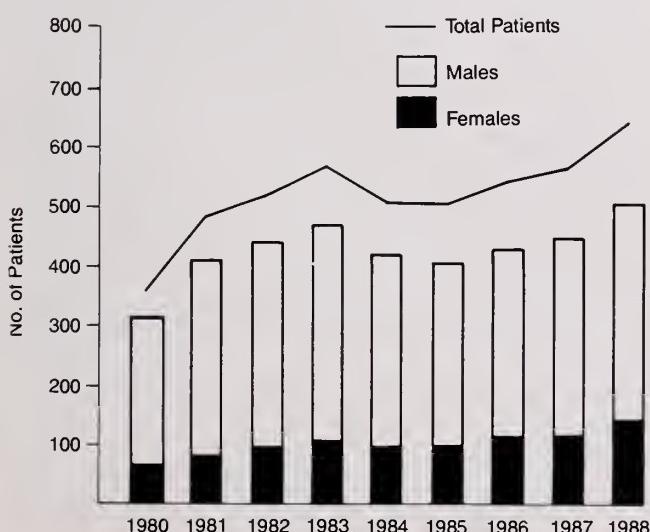
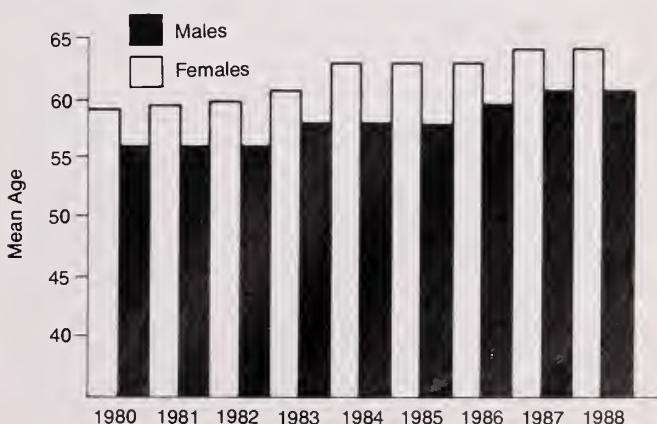


Fig. 2. Mean Age at Operation by Year.



Conclusion

We conclude that patients undergoing coronary artery bypass surgery in the current era differ markedly from those operated upon in earlier years. Barring a dramatic, unforeseen breakthrough in the management of patients with coronary artery disease, we anticipate slowly increasing mortality associated with coronary artery bypass surgery. Earlier studies comparing medical and surgical therapy must be reconsidered in the light of a changing surgical population. Additionally, stratification by risk factors in analyzing coronary artery bypass mortality will become even more critical.

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Figs. 3A and 3B. Risk Factor Incidence by Year.

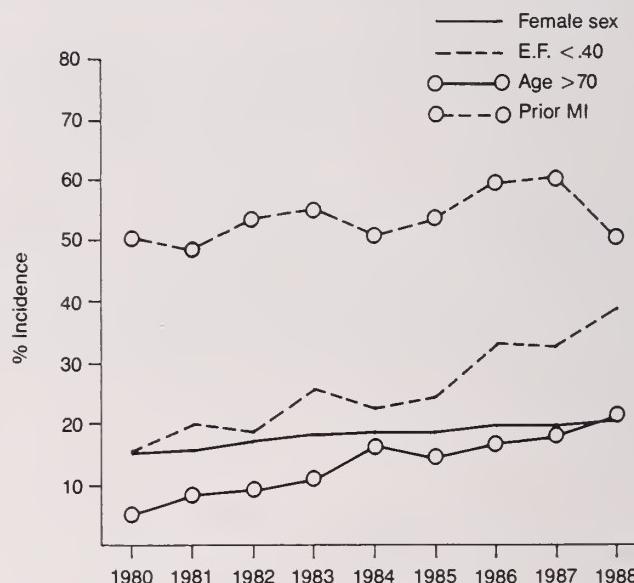
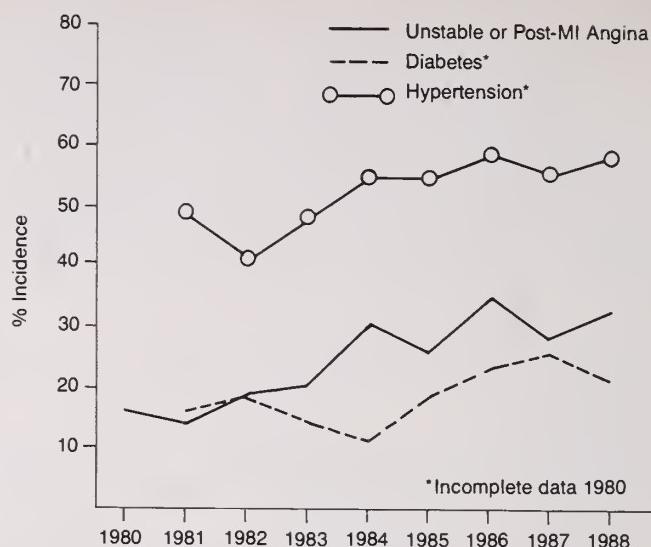
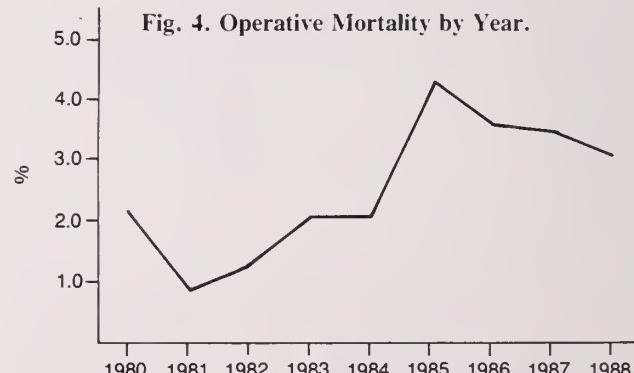


Fig. 4. Operative Mortality by Year.



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Precertification: *Don't Let It Hurt Your Bottom Line*

"In the final analysis, maintaining a smooth and efficient flow of information is the best way to meet precertification requirements—and to minimize the financial and patient-relation repercussions of not meeting them."

In the new reality of medical reimbursement, precertification is a must for most insurance companies—they'll pay for your patients' hospital stays and surgical procedures, on certain specific conditions. The precertification process has been adopted by insurers to control costs and eliminate "unnecessary" admissions. From the patients' perspective, there is nothing wrong with the process, because relatively few are denied coverage, while the process can reduce insurance costs to themselves or their employer.

However, precertification can have a significant impact on your practice's bottom line.

Don't Ignore Requirements

Your failure to obtain precertification—or to discharge a patient within the time period approved by the carrier without getting a "precertified" extension—can result in a lack of payment for the hospital admission or procedure. In that case, responsibility for payment then shifts to the patient.

The first problem that arises here is patient goodwill, which you may lose due to an oversight. The second problem is one of time and personnel management. Claims denied due to precertification problems increase the burden on your office collection process. Precious time, and the extra efforts of your billing staff, will be lost in billing the insurer, researching and challenging the denial, then

rebilling the patient and explaining the reason for the denial. The results will show up on your bottom line; you will see increased staffing costs and a larger portion of outstanding accounts receivable.

Too often, the cost is larger and more directly felt. We have seen situations where physicians have kept patients in the hospital longer than the insurer approved and were left to write off their further charges because the patient could not pay. In one notable instance, a practice failed in its responsibility to obtain the necessary certifications and ended up paying the hospital charges for the "uncertified" days—to assuage an irate patient.

For medical practices, the basic problem with precertification, simply put, is that it is an information-and labor-intensive process. The troubles that develop usually derive from physicians' unfamiliarity with insurers' policies on hospital stays or their procedures for extending those stays. The chart published by VIRGINIA MEDICAL in this issue can help with this problem, but it is not a

long-term solution, since it is subject to short-term change.

Therefore, because of the significant financial effects, practices must do more than review the chart; they must fully understand the general intent and specific points of insurer requirements, make sure their staff does likewise, and develop systems and routines to manage the process.

Gather the Information

Begin to manage the precertification process, rather than allowing it to manage you. Some preliminary legwork can be a real time- and money-saver when scheduling patients for surgery. Have your office manager or billing coordinator make a list of your major third-party reimbursers. Then have your staff contact those carriers and inquire as to their precertification requirements. VIRGINIA MEDICAL's chart provides your staff with the basic information you should request. Here are a few additional thoughts:

- Get answers that are as detailed as possible—exactly what procedures require precertification—and, when possible, have the carriers send the information in writing. Sending a letter to the carrier will usually spur a written reply; you can develop a form letter to standardize the process. Ask the carrier to put you on any regular mailing list it maintains for distributing precertification information and updates.
- Where possible, get the name of a specific individual to contact with

This article was developed by The Health Care Group © 1990. It was written by Patricia Salmon, vice president and senior consultant, and Julie Alderfer, practice management consultant. Address correspondence to The Health Care Group, Meetinghouse Business Center, 140 West Germantown Pike, Suite 200, Plymouth Meeting PA 19462.

your precertification requests and the name of an individual to contact with case-by-case questions. Then list all pertinent phone numbers in your files.

- Note where and when only the physician may request precertification—this will save the staff a phone call. Also note which carriers require that only the patient make the request.
- Detail the procedure to be followed when postoperative complications or other issues require treatment and inpatient time beyond that initially certified by the carrier.
- Determine under what circumstances precertification could be denied and work to avoid those situations.

Organize the Information

As the information flows in, create a reference board that lists all your major carriers' precertification requirements. We find that the best way to keep this information is on an eraseable-marker bulletin board, hung in the business office, or wherever the staffer responsible for precertification is located.

To be sure you have investigated all carriers' requirements, keep a list of those without precertification requirements adjacent to the board. This will probably be a short list. As patients introduce you to additional carriers, you should add those new carriers' information to the reference board.

Your staff should get into the habit of verifying the information with carriers quarterly. As the data changes, the reference board should be updated. As an alternative—or as a backup should the board be accidentally erased—a 3 × 5 card file can be developed. Each card would contain the complete set of precertification information for a single carrier; they would then be filed alphabetically.

Train the Staff

Whenever possible, assign one person the responsibility for coordinating precertification activities. The process and requirements are often so complicated that they necessitate

consistent involvement by one person—a person who will keep abreast of each carrier's requirements and each patient's approval for treatment. This staffer's duties should also include surgical scheduling, because of the natural tie-in to precertification, and we recommend a medical background, an RN or LPN, for example.

However, each staff member should be made aware of the implications of not meeting precertification requirements—delays in surgery and in payments to the practice. Since you will probably see some patients with insurance that is not from one of the "major" carriers listed on your board, your staff must be trained to request and review patients' insurance cards, to identify precertification requirements before those patients are scheduled for procedures. Many patients do not realize that their insurance has such a requirement and will not be able to volunteer the information.

In most cases, it will be your receptionist who will have the best opportunity to get this information, when patients arrive for their first visit. However, the billing coordinator and the person staffing the "exit" desk should also be trained to check for the appropriate insurance and precertification information. In any case, that information should be in the patient's record when the pre-cert/surgery coordinator receives the file and the doctor's directive to schedule a procedure.

If it is not currently being done in your office, the receptionist should make a copy of the front and back of all patient insurance cards and staple that copy to the chart jacket. This information should be verified and updated on a regular basis. Then, if your practice is computerized, a special notation that precertification requirements exist should be made on the patient's insurance screen. If your practice is not computerized, the notation should be made on the patient's ledger card.

Tracking/Updating Certification

We recommend to practices that

they develop a simple form for the precertification coordinator to complete when requesting and receiving clearance for a patient's procedure. This will assure that the correct information has been obtained. The form should be kept in the patient's chart and should include:

- patient's name
- date precertification was requested
- diagnosis
- procedure to be performed
- number of hospital days approved
- precertification approval code
- insurance staffer who approved admission
- hospital staffer who took precert information at time procedure was scheduled
- whether additional hospital days were requested, why, and if approved

Conclusion: Information Is Key

In the final analysis, maintaining a smooth and efficient flow of information is the best way to meet precertification requirements—and to minimize the financial and patient-relation repercussions of *not* meeting them.

The physician plays a key role in this process: first, by identifying precertification as an important issue for the practice; second, by working with the staff to establish a formal precertification routine; third, by recording complete and specific information on diagnoses, procedures, and developing complications—which allows the staff to carry out its responsibilities quickly and effectively.

Perhaps though, the most important part of this information loop—where the process begins and ends—is the individual patient. You must educate your patients on the purpose and widespread use of precertification requirements, and on the potential dollar-impact for the patient. Your effort to educate patients can help prevent problems if they feel they are being discharged too soon. Such education can be accomplished through your patient-education materials, your practice newsletter, or during pre-surgical counseling.

VIRGINIA MEDICAL'S Guide to Precertification in Virginia

Virginia's hospitals racked up an estimated 720,000 admissions in 1989,¹ and every last one of those patients was admitted by a Virginia physician. Until recently, arranging for a patient's hospitalization in Virginia was no big deal. In the normal circumstance all a doctor had to do was call up the hospital and reserve a bed.

But in 1984 precertification of hospital admissions was imposed in Virginia, and since then doctors have not only had to work up their patients, a clinical task they love, but they have had to work up proof that the patient's hospital admission is necessary, an assignment that went instantly to the top of the doctors' regulatory hate list.

Precertification, or preauthorization, as some call it, is a manifestation of the brave new world of managed medical care. Its intended goal is 1) to discipline medical costs so that 2) insurance premiums, already at unseemly levels, don't break everybody's budget. What better place to implement it than in the physician's office, where any unnecessary hospital admissions can be nipped in the bud?

The goal is worthy, the approach problematic:

Physicians have not been used to having their treatment plans nipped, in the bud or anywhere else. And they have not been accustomed to shuffling umpteen papers to prove the need for a patient's hospitalization. And they were absolutely appalled when they were presented with the laborious, time-consuming precertification process of pleading each case, one by one, according to the variable criteria of myriad plans/carriers/review agencies.

In the beginning, five years ago, the variables were indeed a nightmare. One plan didn't know what the other was doing. Subscribers' identification cards were mazes of fine print. Phone lines seemed infernally busy or didn't answer.

Today, thanks to national standards, VIRGINIA MEDICAL's chart demonstrates a much simpler, almost codified, system.

Simpler, yes, but simple it still isn't. Despite valiant efforts by plans and carriers and reviewers to beef up their answering powers with more phones and savvy personnel, the physician may still encounter road blocks—lots of ON HOLD, lots of delays because the screening person isn't sufficiently knowledgeable, lots of non-callbacks, lots of patient demographics required. Doctors perceive such hangups as barriers to patient care, and barriers to patient care profoundly disturb even the most fervent advocate of managed medicine. A Richmond physician who is such an advocate says that if he thinks something is really wrong with the way his precertification request is handled, he fires off a letter of complaint to the State Insurance Commissioner. It helps him a lot to get it off his chest. More importantly, he hopes it will lead to improvements in the system. But whether it does or not, this, he believes, is the bottom line:

"Preadmission certification is a fact of life, and precertified ambulatory surgery is on the way in. We've got to learn how to operate the system."

The plans/carriers/review agencies who graciously contributed data to this chart precertify most of the hospital admissions in Virginia. Their information is subject to change, but this bird's-eye view of certification, assembled for the first time by VIRGINIA MEDICAL, may serve as a lasting context for the Virginia physician as he goes about getting his patients into the hospital.

—ANN GRAY

1. Estimate based on projected figures, Virginia Hospital Association, January 24, 1990

Name/Address of Plan	Aetna Life and Casualty 2809 Emerywood Parkway Richmond VA 23260	Blue Cross Blue Shield of Virginia 2015 Staples Mill Road PO Box 27401 Richmond VA 23279
Reviewer Name/Address	Same as above	Blue Cross Blue Shield of Virginia Health Care Management PO Box C32104 Richmond VA 23261-2104
Certification Phone Number	See member's ID card	If patient's contract ID specifies "Admission Review Required," call 804-359-7277 or 1-800-242-7277.
What procedures require precertification for medical indications?	See ID card	Premark authorization not required, but subscriber may request it for high-dollar services, transplant, growth hormones, potentially cosmetic/investigative procedure, etc.
What procedures require precertification for setting?	See ID card	Contract specific. Most group contracts require review of all hospitalizations.
Who may request certification?	Patient, MD office, hospital admissions	Physician, patient, hospital
Who handles certification requests?	Physician reviewer, RN	RNs, physicians
What data will be asked for?	Patient: name, address, birthdate, SS#, ID#, relationship to insured person, company name. Physician: name, address, phone, tax ID#. Procedure: location of service, admission date, estimated length of stay, treatment plan.	For Admission Review Program: diagnosis, treatment, lab values, history, other medical conditions. All others: diagnosis, functional problems, indications for surgery, procedures/services required.
When must preadmission certification be obtained?	14 days prior to elective admission (or as soon as admission is scheduled)	Before hospital admission. Maternity admission in last trimester.
How about an emergency?	Within 48 business hrs after admission	Within 48 hours
Who issues denials?	Usually by physician, sometimes RN	Nurse reviewer, medical director
How can a denial be appealed?	Request reconsideration. Case then reviewed by another physician reviewer or medical director.	Submit written request for appeal with medical documentation
If MD violates denial, what is penalty?	Partial or complete denial may occur	Claim can be denied
To ask question or make complaint	Aetna toll free # on ID card	Donna Campbell, RN 804-342-2668
Medical director	Richard Bondi, MD	Richardson Grinnan, MD, Chief Healthcare Officer

CHAMPUS	Choice Health Plan 8550 Lee Highway Fairfax VA 22031	George Washington University Health Plan 1901 Pennsylvania Avenue NW Washington DC 20006	Group Health Association 4301 Connecticut Avenue Washington DC 20008	HMO Virginia 2220 Edward Holland Drive Richmond VA 23261	Kaiser Permanente Medical Program of the Mid-Atlantic States 4200 Wisconsin Avenue NW Washington DC 20016
Medical Society of Virginia Review Organization PO Box K70 Richmond VA 23288	Same as above	Same as above	Robert Younes, MD, Regional Medical Director, GHA 5109 Leesburg Pike Falls Church VA 22041	Same as above	Same as above
1-800-533-1745, 804-289-5336 Fax: 804-289-5395	703-849-9772	202-416-0477	703-824-3005	Richmond: 804-355-6327 Tri-Cities: 1-800-533-5592 Tidewater: 1-800-533-5592	1-800-765-2262
Tonsillectomy/adenoidectomy (single or combined), cholecystectomy, hysterectomy, C-section	All hospital care, surgeries, procedures outside MD office, services by non-participating providers	Mammoplasty (reduction, augmentation), septoplasty/rhinoplasty, temporal mandibular joint syndrome surgery, nevus removal, varicose vein surgery, circumcision, blepharoplasty	All procedures	All procedures done in hospital or ambulatory surgery setting	All care rendered by Kaiser Permanente physicians
None. All considered inpatient.	All procedures	Inguinal hernia repair, cataract extraction, oral surgery, endoscopy, arthroscopy	All procedures	All procedures	NA
Physician or designated representative	Primary care physician, staff, specialist	Physician, nurse, secretary, office workers	Physician or staff	Physician or staff	NA
MSVRO RNs, physician reviewers	RNs supported by medical director	RN. May refer to medical director.	Primary care physician	PCP must approve all admissions	NA
Patient: name, address, birthdate, relationship to insured; insured's name, address, SS#. Physician: license #. Procedure: diagnosis, hospital, admission date, unstable medical condition(s) affecting surgery.	Patient: name, birth date, ID #. Procedure: diagnosis, IP or OP, location of service, admission/procedure date, supporting clinical data (prefer written).	Patient: name, home/work phone, birthdate, Plan medical record #. Physician: name, phone, specialty. procedure: diagnosis, location of service, procedure date, anesthesia, referral # if available.	Patient: name, ID#. Physician: name. Procedure: diagnosis, IP or OP, admission date.	Patient: name, ID #, name of PCP. Physician: name. Procedure: diagnosis, location of service, admission date.	NA
72 hours prior for phone or fax, 10 days prior for mail-in. Certification valid for 60 days from issue.	Sufficiently prior to admission for second opinion if needed	At least 1 week prior to procedure	One week	At least 24 hours. OB patients within 30 days of due date.	NA
Precertification required. Can be faxed prior to procedure or phoned in within 2 working days of procedure.	PCP must authorize before services rendered. In life-threatening situation, PCP or member must notify Plan within 24 working hrs.	Call before procedure if possible. Or retrospective authorization may be given.	Within 24 hours	As soon as possible	Notify Kaiser Permanente at time of treatment (1-800-765-2262). Patient must file claim.
MSVRO physician reviewers	Medical director	Medical director	Primary care physician, regional medical director, claims processing unit	PCP and Plan director	Kaiser Permanente
MD or patient can ask for immediate reconsideration of denial. Case thoroughly re-reviewed by another physician.	By phoning/writing medical director	MD may contact medical director for reconsideration of denial	Write Claims Management Unit 4301 Connecticut Avenue Washington DC 20008	Formal grievance process (see manual)	Patient appeals by procedure outlined in handbook
Claim may be denied	Claim may be denied	Plan reimburses only precertified procedures, except emergencies	Claim will be denied	Payment will be denied	NA
Cynthia Payne, RN 804-289-5320	Member services, 1-800-537-5096	Christina Bridgeport, RN 202-416-0477	Robert P. Younes, MD 703-824-3005	Richmond: 804-358-9088 Tri-Cities: 1-800-421-1883 Tidewater: 1-800-421-1883	1-800-765-2262
Eugene F. Poutasse, MD	Edward J. Smith, MD	Neeraj Kanwal, MD	Turner Bledsoe, MD	W. Richard Stubbs, MD	Martin Bannan, MD

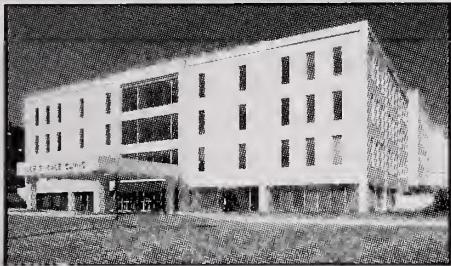
Name/Address of Plan	Medicaid 600 East Broad Street Richmond VA 23219	Medicare	Partners Health Plan of the Mid-Atlantic 8550 Lee Highway Fairfax VA 22031	Southern Health Services 4222 Cox Road Glen Allen VA 23061
Reviewer Name/Address	State Department of Medical Assistance Services	Medical Society of Virginia Review Organization PO Box K70 Richmond VA 23288	Same as above	Same as above
Certification Phone Number	804-786-3820	1-800-533-1745, 804-289-5336 Fax: 804-289-5395	1-800-468-7730 or 703-876-2855	804-270-9200 Tri-Cities: 804-748-9240 Nights/weekends: 804-312-0409
What procedures require precertification for medical indications?	Elective surgery, i.e., morbid obesity, vaginal breast hypertrophy	Cataract extractions, carotid endarterectomies, cholecystectomy, major joint replacement, coronary artery bypass with graft, percutaneous transluminal coronary angioplasty (PTCA), laminectomy for herniated lumbar discs (L2-S1), hysterectomy (abdominal and vaginal), inguinal hernia, prostatectomy	All hospital care, surgeries, procedures outside MD office, services by non-participating providers	Procedures/services listed in <i>Evidence of Coverage and Physician Office Manual</i> . Call for details.
What procedures require precertification for setting?	See Medicaid physician's manual; App B, page 1 thru 6	Cataract extractions, inguinal hernia	All procedures	See above.
Who may request certification?	Physician	Physician or designated representative	Primary care physician or specialist	Physician
Who handles certification requests?	Physician reviewer	MSVRO RNs, physician reviewers	RNs	RNs
What data will be asked for?	Patient: name, address, birthdate, sex, Medicaid #. Physician: name, address, phone, Medicaid provider #. Procedure: location, IP or OP, date of procedure, diagnosis, procedure, medical indications.	Patient: name, address, birthdate, sex, Medicare #. Physician/surgical assistant: name, address, license #. Procedure: diagnosis, IP or OP, location of service, admission date, indications, unstable medical condition(s).	Patient: name, birthdate, ID#. Procedure: diagnosis, IP or OP, location of service, admission/procedure date, supporting clinical data (prefer written).	Patient: name, ID#. Physician: name. Procedure: Date admission, date service, hospital/facility, diagnosis, procedure/treatment/services.
When must preadmission certification be obtained?	Minimum 48 hrs prior to surgery	72 hours prior for phone or fax, 10 days prior for mail-in. Certification valid 60 days from issue.	Sufficiently prior for review/second opinion (recommend 7-10 days before services rendered)	Prior to procedure
How about an emergency?	Not necessary. Emergencies covered if justified by documentation.	Precertification required. Can be faxed prior to procedure or phoned in within 2 working days of procedure.	PCP must authorize. If life-threatening situation, PCP must notify Plan within 24 working hours.	Within 24 hours, not including weekends and holidays
Who issues denials?	Physician reviewer	MSVRO physician reviewers	Medical director	RNs. All denials reviewed by nurse manager and medical director.
How can a denial be appealed?	Physician may request reconsideration. Case given to another physician reviewer for 2nd evaluation.	MD or patient can ask for immediate reconsideration of denial. Case thoroughly re-reviewed by another physician.	By phoning/writing medical director	Phone nurse manager or medical director. Written appeal to Utilization Review Committee.
If MD violates denial, what is penalty?	Payment withheld or adjusted	Claim subject to denial	Claim may be denied	Payment will be denied
To ask question or make complaint	Helpline for billing/general info: Richmond area, 804-786-6273 Other areas, 1-800-552-8627	Cynthia Payne, RN 804-289-5320	Member services, 1-800-537-5096	Member services: 804-270-7800 Provider services: 804-747-3700
Medical director	Robert L. Wood, MD	Eugene F. Poutasse, MD	Edward J. Smith, MD	Mark A. H. Smith, Jr., MD

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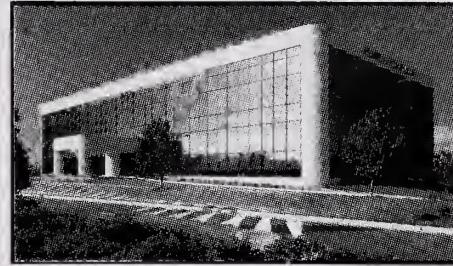
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Biliary Obstruction and Cholestasis in AIDS: Case Report

Sheldon M. Markowitz, MD,
Thomas M. Kerkering, MD, and
Alfred S. Gervin, MD, Richmond

PERSONS infected with the human immunodeficiency virus type 1 (HIV-1) frequently develop gastrointestinal symptoms and signs, including weight loss and diarrhea. A number of recent reports have documented the involvement of the biliary tract in the acquired immunodeficiency syndrome (AIDS).¹⁻⁷ Most patients have presented with a clinical, biochemical and radiological picture of biliary obstruction, usually associated with varying degrees of stenosis of the intrahepatic and extrahepatic biliary systems. Gallstones universally have been absent. Cytomegalovirus, cryptosporidia and other as yet unidentified pathogens have been proposed as likely etiologic agents, leading to edema, inflammation and fibrosis of bile ducts. We describe herein a previously unrecognized cause of biliary tract obstruction and cholestasis in a patient with AIDS.

Case Report

A 32-year-old female with a diagnosis of AIDS had been treated previously for pulmonary and disseminated cryptococcosis and disseminated *Mycobacterium avium-intracellulare* infection. She had received approximately 2 grams of amphotericin B and was being maintained on weekly amphotericin B and daily rifabutin, clofazimine, ethambutol and isoniazid. She was hospitalized with a one-week history of nausea, vomiting, fever and right upper quadrant abdominal pain. On physical examination, her temperature was 38.6°C orally. Epigastric and right upper quadrant abdominal tenderness and Murphy's sign were present. The gallbladder was palpable. Laboratory examination revealed the following: total bilirubin, 0.2 mg/dl; alkaline phosphatase, 368 U/L (normal, 20-125); lactate dehydrogenase, 192 U/L (normal, 100-125); gamma-glutamyl-transpeptidase, 170 U/L (normal, 0-45); alanine aminotransferase, 36 U/L (normal, 0-50);

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Submitted 5-11-89.

serum creatinine, 1 mg/dl; and serum amylase, 145 U/L (normal, 16-108). Two-dimensional ultrasonography of the right upper quadrant revealed a distended, thin-walled gallbladder with "sludge" but no gallstones (Fig. 1A).

Because of progressive and intractable symptoms, the patient underwent a right upper quadrant abdominal exploration two days after admission. At operation, the gallbladder was noted to be markedly distended. The common duct was normal in size without obstruction. However, the cystic duct was dilated and found to be obstructed by multiple enlarged periductal lymph nodes. Multiple nodes were biopsied for histopathology and cultures and the obstructing nodes excised. A cholecystojejunostomy was performed.

The patient's symptoms abated rapidly following surgery, while the biochemical pattern of biliary obstruction worsened in the immediate postoperative period, only to improve later. Histologic examination revealed the virtual replacement of normal nodal architecture by histiocytes containing organisms compatible with cryptococci (Fig. 1B). No viral inclusions were seen. *Cryptococcus neoformans* was isolated from cultures of the lymph nodes.

One month following surgery, repeat two-dimensional ultrasonography of the right upper abdominal quadrant revealed a slightly distended gallbladder. On examination, abdominal tenderness was absent and her gallbladder was no longer palpable. Liver function studies were markedly improved. Three months following surgery, the patient developed *Pneumocystis carinii* pneumonia and acute myocarditis and later died of a recalcitrant ventricular arrhythmia. No autopsy was obtained.

Discussion

Abdominal symptoms and signs in patients with AIDS may result from multiple visceral organ involvement by opportunistic pathogens and by various tumors.⁶ A change in bowel habits, weight loss and diarrhea are common, occurring in 50% or more of HIV-infected individuals.² The incidence of abdominal pain, particularly as an early manifestation of HIV infection, is not known, but abdominal pain has been associated with pathology at all levels of the gastrointestinal tract, including esophagitis, gastroduodenitis, enterocolitis, proctitis, hepatitis and hepatosplenomegaly, pancreatitis, peritonitis, retroperitoneal lymphadenopathy, gastrointestinal bleeding, and intestinal perforation.^{2,8} At least 26 patients with AIDS and significant gallbladder disease have been reported since 1983.¹⁻⁷ Typically, patients presented with symptoms, signs and biochemical evidence of cholestasis and biliary obstruction, often with marked elevation of the alkaline phosphatase and lesser degrees of elevation of aminotransferases. Over half had normal serum bilirubin levels, including all patients reported by Schneiderman.⁶ Gallstones were absent in all in-

stances, although there was presence of sludge in a few. Characteristic findings in most patients were structural abnormalities of intrahepatic and extrahepatic bile ducts associated with infection by cytomegalovirus, cryptosporidia, *Candida albicans* and *M avium-intracellulare*.¹⁻⁷ In a recent report of AIDS cholangiolopathy,⁷ six of 26 patients with clinical and laboratory evidence of biliary tract disease were found to have normal retrograde cholangiograms and no histopathologic abnormalities on biopsy of the duodenal papilla. *M avium-intracellulare* was isolated from the blood cultures of one patient and the liver biopsy of another, but for these patients, as for all those previously described, no causal relationship was established.

The clinical presentation of this patient was similar to others reported, except for the presence of a palpable and distended gallbladder, later confirmed by right upper quadrant abdominal ultrasound. The findings of an enlarged and distended gallbladder essentially eliminated choledocholithiasis as a cause (Courvoisier's law) and, with evidence of a common duct of normal caliber, pointed to cystic duct obstruction.

The explanation for the otherwise characteristic presentation of cholestasis and biliary colic in this patient with AIDS was found on surgical exploration. Cystic duct compression by enlarged periductal lymph nodes was noted. Cryptococcal lymphadenitis was later conformed by histopathologic examination and culture. The association of cryptococcal visceral lymphadenitis and cystic duct obstruction has not been described previously. Of 61 patients with cryptococcal disease and AIDS, none were found to have biliary tract disease and only one, in fact, had abdominal pain.^{8,9} Although concomitant cytomegalovirus or cryptosporidial biliary tract infection could not be excluded, the absence of common duct abnormalities on ultrasound and the normal appearance of bile duct radicals on a liver biopsy suggested that the structural changes seen in other such patients were not prominent in this individual, at least at the time of surgery.

The difficulty in treating disseminated cryptococcosis in patients with AIDS was apparent in this patient. Despite having received over 2 grams of amphotericin B, followed by weekly maintenance doses, progressive cystic duct obstruction due to cryptococcal lymphadenitis occurred. Interestingly, the patient's serum liver function profile worsened following biliary decompression, while she clinically improved. This phenomenon was observed in four of eight patients described by Schneiderman et al and was considered a poor prognostic sign, as two of these individuals later developed biliary sepsis and required further biliary decompression.⁶

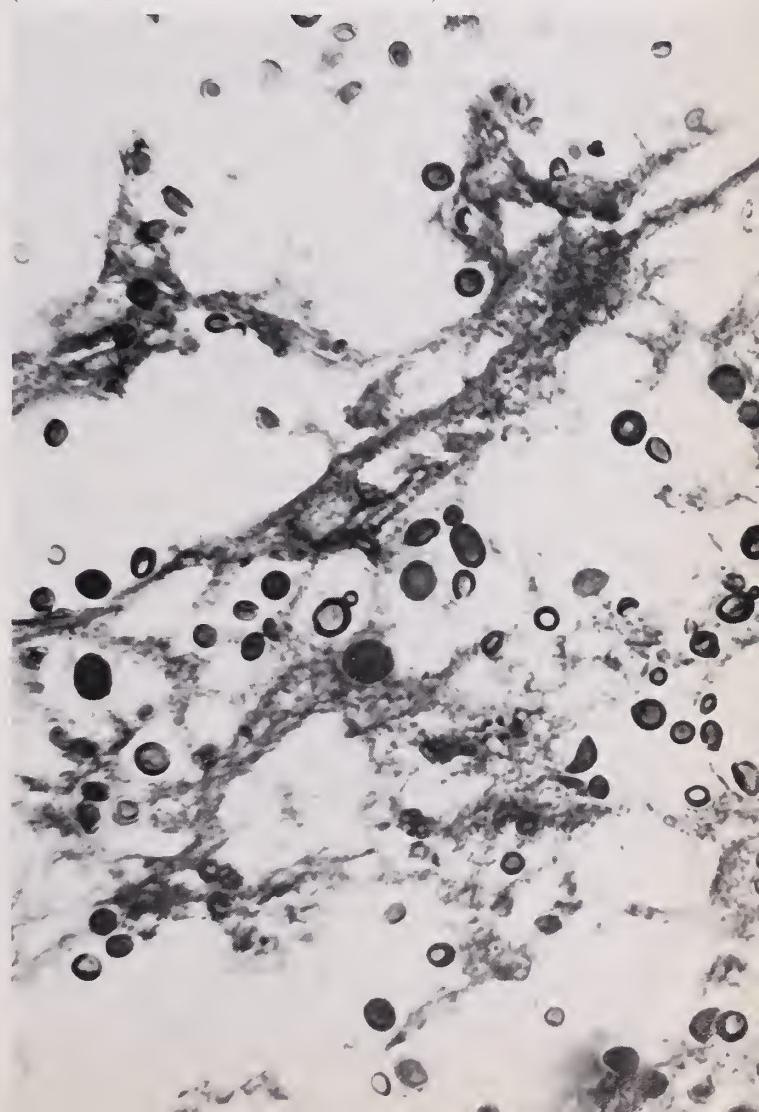
Summary

In summary, we have described a patient with AIDS and a previously unreported cause of biliary tract



Fig 1A. Two-dimensional ultrasound study showing enlarged, thin-walled gallbladder without stones.

Fig 1B. Lymph node containing multiple encapsulated budding yeast cells, compatible with *Cryptococcus neoformans* (Gomori methenamine silver stain $\times 320$).



obstruction. The incidence of cryptococcal visceral lymphadenitis in patients with AIDS and disseminated cryptococcosis is unknown, but, if present, is probably clinically silent in most instances. However, in the differential diagnosis of abdominal pain and cholestasis in such patients, one should consider major biliary duct obstruction due to cryptococcal lymphadenitis.

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Tinea Nigra Palmaris in Virginia: Case Report

Samuel T. Selden, MD,
Chesapeake, Virginia

TINEA nigra palmaris is a heavily pigmented, superficial fungal infection, usually involving the palm of a single hand. It is uncommon outside the tropics. I report a case in a Virginia girl, treated with econazole and tretinoin creams.

Case Report

An 8-year-old white girl developed a brown stain on her right palm over the course of 18 months. There had been no scaling, itching or vesiculation. The girl had spent her entire life in Virginia and had never visited beyond the Commonwealth's borders. She denied any chemical exposure or injury.

On examination there was a 25 × 30 mm dark brown stain in the center of her right palm. No elevation or scaling was evident. There was a slight clearing of the lesion's center. Although it appeared as a stain or as dirt, a brisk rubbing with alcohol had no effect on the lesion. The girl's left palm and the plantar surface of her feet were free of lesions.

A scraping was done from the periphery of the lesion, and on potassium hydroxide (KOH) testing, hyphae were visible. A fungal culture prepared on Mycosel's medium eventually produced a grey fuzzy growth, consistent with *Exophiala wernickii*.¹

Treatment was begun with a twice daily application of 0.01% tretinoin gel and 1% econazole nitrate cream. In two weeks the pigment had faded appreciably. At one month the palm was completely clear and treatment was stopped. Six months later there had been no recurrence.

Discussion

E wernickii infection occurs after penetration of the stratum corneum by the fungus. Over time, a spreading dark stain develops, due to release of fungal pigment. This pigment is superficial and can be scraped or shaved off, differentiating this condition from nevi or melanomas, where the pigment is deeper and impervious to shaving.²

Pigmentation of the palm could also be caused by dirt, chemical staining, postinflammatory hyperpigmentation, or melanocytic lesions. In this case, the

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positive KOH test and culture effectively ruled out the other causes.

Tretinoin (used as a keratolytic) and econazole successfully cleared the infection. Whitfield's ointment, with salicylic acid as the keratolytic, has been cited as effective in the past. In this case, tretinoin served to strip away the thick palmar stratum corneum to better expose the fungus and to allow more effective penetration of the econazole.

Although not listed as effective against *Exophiala wernickii* in vitro, exonazole, as a new generation antifungal, might be expected to work. Miconazole has been used in the past,³ but to my knowledge no

reference has yet been made in the literature as to the effectiveness of the newer antifungals against tinea nigra.

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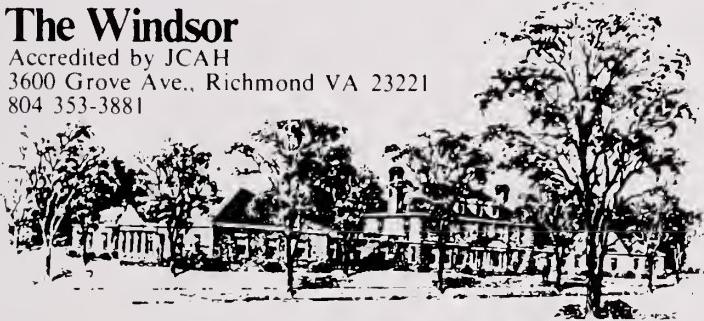
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Changing Perspectives: Coronary Artery Disease

CORONARY artery bypass surgery continues to impact significantly on the management of patients with coronary artery disease. While symptomatic benefits of surgery have long been recognized, defining longevity benefits has been slower. Decisions regarding appropriate patient selection for specific therapeutic management is clearer today. For example, factors predicting the most demonstrable survival benefits of bypass surgery include the extent of coronary artery disease, the extent of left ventricular dysfunction and the severity of demonstrable ischemia. Physicians are more appropriately attuned to these factors and as a consequence the patient population referred for surgery is changing, i.e., patients with poor left ventricular function, worse coronary disease, and even the elderly are now commonplace. As a consequence, mortality has increased but comparative studies of medical versus surgical therapy have demonstrated that certain high-risk patient groups, particularly those having left ventricular impairment with more extensive coronary disease¹ or severe ischemia, such as unstable angina,² experience improved cumulative yearly survival following bypass surgery.

In addition, the elderly can also benefit from surgical revascularization, though at a slightly increased risk.³ The importance for the elderly may actually be greater, as myocardial hypertrophy, which appears with age, seems to decrease these patients' tolerance to ischemic events, thereby increasing the potential impact of revascularization.

Thus, as a consequence of these findings, a changing

coronary surgical population is occurring, as pointed out by McManus and colleagues in this issue of VIRGINIA MEDICAL. However, the late outcome for appropriately selected high-risk patients is nonetheless enhanced. This message must be transmitted to review organizations and to the public: Treatment outcomes are clearly dependent upon patient selection, but selecting appropriate high-risk patients may ultimately improve outcome.

Finally, coronary angioplasty has reduced the number of patients with one- and sometimes two-vessel disease undergoing bypass surgery for medically uncontrolled symptoms with quite acceptable morbidity and mortality results.⁴ Multiple randomized trials comparing surgery versus angioplasty for more extensive disease are ongoing and should help to again better define those populations most likely to benefit from bypass surgery.

Decision-making and technology continue to evolve. The challenge remains to appropriately select patients who may potentially achieve the greatest survival and symptomatic benefits from our therapeutic decisions.

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Medical School Applications

ACCORDING to the Association of American Medical Colleges (AMA News, 17 November 1989), the 14-year decline in applications to medical schools may have bottomed out. On the basis of applications received by the first week of November 1989, the AAMC estimates that the number of applicants for admission to medical schools next year will top the 1989 applications by about 1,000. Virginia medical schools report a similar response.

Applications to the Medical College of Virginia/VCU are up by 25%; Eastern Virginia Medical School has experienced a 30% increase; and 11.2% more applications have been received by the University of Virginia School of Medicine.

The reasons for this increased interest in medicine? The AAMC suggests that the increase in applications may be due to the current ratio of 1.6 applicants for each medical school slot, an historic low. However, Dr. Robert Beran, assistant vice president, AAMC, notes that all other health professional schools also have experienced an increase in applications.

Admissions officers of the three Virginia medical

schools likewise are unable to provide a specific answer to the increased number of applications. In Norfolk, Dr. Robert McCombs suggests that EVMS recruiting and increased interest in out-of-state students may be a major factor. Further, the school has become more widely known.

Dr. Edwin Pullen, University of Virginia dean of admissions, notes two factors: more active recruiting by medical schools and the perception of instability in business, as evidenced by the 1987 stock market crash.

At MCV, Dr. Kenneth Blaylock agrees that the greater likelihood of admission (1.6 ratio of applicants to medical school slots) has activated more candidates. He further agrees with Dr. Pullen that medicine is more widely perceived as a desirable profession.

Other factors in the trend, as noted by the AAMC and seconded by Dr. Blaylock, appear to be a rise in applications from women and minorities, particularly Asian Americans. On the other hand, there has been a decrease in applications by white males.

E.L.K., JR.

Great Communication

I KNEW her well, both socially and professionally, and I liked her. After she began to develop cataracts, I made more than my usual effort to explain the advancing problem to her.

Her lens opacities slowly grew worse. Finally new glasses would not improve her visual disability, and cataract surgery was obviously necessary.

I explained why new glasses would, again, not help and why only cataract removal offered her a chance for improved eyesight. With a model eye I went into detail about what cataract surgery entailed.

She wanted to think it over and returned in a week with a few questions which I answered with great skill and clarity. Just to be absolutely certain that she

understood completely, I again gave a superlative explanation with the model eye. She obviously understood by her facial expression and by her having no more questions. So we scheduled her surgery.

She returned to see my partner the next day to have ultrasound measurement of her eye in order to calculate the power of the planned intraocular lens implant. After that determination was completed, she and my partner chatted.

Just as she was preparing to leave, she turned to my partner and said, "Don, please answer some questions for me. Henry never tells me anything."

H.S. CAMPBELL, MD

VIRGINIA MEDICAL OBITUARY

- **Sterling Neblett Ransome, MD**, Mathews, a past president of the Mid-Tidewater Medical Society; Medical College of Virginia, 1956; age 63; died January 5, 1990. Dr. Ransome retired from practice in 1989.
- **Douglas Doriot Vance, MD**, longtime family physician in Bristol, Tennessee; University of Virginia School of Medicine, 1928; age 93; died April 14, 1989.
- **Lewis Emmor Wells, MD**, retired Farmville internist; University of Pittsburgh School of Medicine, 1931; age 83; died October 31, 1989, in Fayetteville, North Carolina.

Memoir of George Hurt 1889-1989

By Alexander McCausland, MD, and
William Kaufman, MD

It is with a great sense of loss that we present this memorial resolution for our oldest, longest standing member and past president (1935-36) of the Roanoke Valley Academy of Medicine.

Dr. George Samuel Hurt was born in Elizabethtown, Kentucky, December 4, 1889. He died in Roanoke, Virginia, March 28, 1989, at the age of 99.

Dr. Hurt was educated at Fork Union Military Academy and the Medical College of Virginia. He began the practice of medicine in Roanoke in 1913 and continued until he was commissioned in the army July 6, 1917. He helped organize Ambulance Company No. 46, which eventually became Ambulance Company No. 319, a unit of the 80th Infantry Division. On May 1, 1918 the division embarked for France, where upon arrival Dr. Hurt's company was assigned to the British Army, serving at the front until reassigned to the American Army.

On September 15, 1918, his unit was in reserve during the St. Mihiel Offensive. It then moved to an area in the American sector northwest of Verdun. An advance dressing station was set up in preparation for the Meuse-Argonne offensive, which began the morning after arrival. Dr. Hurt continued to follow the advance through all three phases of the offensive. During the winter of 1918-19 while billeted at Pimmeles, France, he started a hospital for his own men which later became the division hospital and which operated as such until the movement began for the

journey home. He refused many opportunities to leave the lines for a rest period and, although severely gassed, remained at his post until his division was relieved two days before the Armistice. For these acts of distinction he was awarded the service cross. The citation read: "At Natillois while maintaining a dressing station under severe shell fire, Lieutenant Hurt was severely gassed, the eyes and face were burned and laryngitis and bronchitis developed, causing a loss of voice for about two weeks and a cough that lasted two months. Regardless of these painful and incapacitating conditions, Lieutenant Hurt refused to be evacuated and not only maintained his own station but was instrumental in establishing and supervising other stations. This officer was one of the most progressive, efficient and tireless workers in Ambulance Company 319 and during the thickest fighting was known to have worked 72 hours without sleep."

Dr. Hurt was discharged May 19, 1919, and returned to Roanoke to resume his medical practice. He was a member of the Disabled Soldiers of America and the American Legion, and was commander of the Roanoke American Legion Post. He was very active in obtaining aid for widows and orphans of former soldiers.

He served many posts in the Calvary Baptist Church: He was a Sunday School teacher, he served on the Board of Finance, he was chairman of the Board of Deacons, superintendent of Sunday School, chairman of the brotherhood, chairman of the Nominating Committee, chairman of the Pastor Procurement Committee and chairman of the Library Committee. He served as a trustee of the church. He contributed also to special missionary undertakings abroad. In 1960 the church honored him by making him deacon emeritus.

Dr. Hurt was a member and president of the Roanoke Academy of Medicine (1935-36), member of the Virginia Association of Gynecologists and Obstetricians, the Roanoke Association of Gynecologists and Obstetricians, and first vice president of The Medical Society of Virginia. He served as president of the YMCA and as a member of the United Fund. During World War II, he was appointed senior surgeon in the United States Public Health Service, serving in that capacity as a consultant. During World War II he served as a member of the Selective Service System for four years, for which he was awarded the Certificate of Merit signed by President Roosevelt. He also received the Certificate of Merit for Distinguished Service to the USO, signed by Truman.

Dr. Hurt married Miss Corinne Lockett May 8,

1918. They had one daughter, Mrs. Corinne Hurt Turner, and three sons. George William Hurt, David Edgar Hurt, and Alvin Judson Hurt. Two of the sons, Drs. George and Alvin Hurt, followed in their father's footsteps in the practice of obstetrics and gynecology in Roanoke. A grandson, George Samuel Hurt III, practices urology in Martinsville, Virginia. There are six grandchildren and seven great-grandchildren.

Dr. Hurt was always immaculately dressed. He never failed to wear a flower, especially a rose, in his lapel. A testing in the fires of World War I was succeeded by a serene maturity and old age, which the flower in the buttonhole symbolized. "And the fire and the rose are one," wrote T. S. Eliot.

The interns, residents and nurses considered Dr. Hurt their favorite physician. The Jefferson Hospital Nurses Alumni Association memorialized Dr. Hurt with a contribution to the Memorial Chapel Fund at Community Hospital of Roanoke Valley.

If friends or members of a doctor's family were in the hospital, Dr. Hurt was the first to pay them an encouraging personal visit. He never missed his daily visits while they were in the hospital. His hospital rounds included not only his patients, but also a good-morning visit to the lab, x-ray, record room, administrative offices and dietary department. If a doctor was needed at the blood bank, he was always willing to serve.

Sunday afternoon found Dr. Hurt making home and personal visits to friends and acquaintances. He was a friend to the young and new doctors, and a wise counselor to all doctors. To the interns and residents he was an inspiration.

With Dr. Hurt's passing our city and our state lost a great and good servant. Medicine has lost a dedicated physician who practiced in keeping with the highest traditions of American medicine. He gave much to medicine and through it, to his fellow men. We who were privileged to know him have lost a warm and loyal friend.

News of the death of a Medical Society of Virginia member may appear in these pages in either or both of two ways: 1) a brief notice written by staff giving factual information; 2) a memoir contributed by a physician who knew the deceased peer and writes a personal account of his or her life. If the memoir is contributed before the brief notice is inserted, it is published at once; if after, it is set in type to be published as space permits.

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References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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Manuscript Preparation

Medical articles, editorials, essays, Letters to the Editor and all other text submitted for publication must be double-spaced throughout, including references, legends and all other elements. The material should be typed on one side of the paper, with generous margins of at least 1 1/4 inches all around. Do not use all-caps or a script typeface. Submit one original of the communication and one copy. If the material is not accepted, the original will be returned; the copy will be retained.

The author is responsible for the accuracy of all statements and references. Acronyms and other abbreviations should be kept to a minimum; unless an acronym is widely known and used by all specialties, it should be fully explained in the text. Refer to pharmaceutical products by their generic names; brand names may follow in parentheses and should carry registered trademarks where applicable. All units of measure should appear in the metric system. References, typed in double-space, should be listed in the order of their citation in the text, not alphabetically. They should follow VIRGINIA MEDICAL's typographic style for references; the typist should study this style as it appears in each issue.

Illustrations should be black and white glossy prints, with legends typed in double-space on a separate sheet of paper. VIRGINIA MEDICAL has no budget for printing in color; the author who wishes to publish a four-color figure may negotiate to pay for the costs.

Attach to the contribution a covering letter giving the address and telephone number of the person who will correspond about it and address the completed communication to the Editors, VIRGINIA MEDICAL, 4205 Dover Road, Richmond VA 23221.

All manuscripts are subject to editorial changes. If extensive revision is deemed necessary, the author will receive for approval a draft of the article as edited.

There are many excellent handbooks of effective writing, among them *The Elements of Style*, by William Strunk, Jr., and E. B. White (MacMillan); *The Careful Writer: A Modern Guide to English Usage*, by Theodore M. Bernstein (Atheneum); and *How to Write and Publish a Scientific Paper*, by Robert A. Day (ISI Press).

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MEETINGS

April 1-4

Conference on AIDS (Johns Hopkins), *Baltimore*. Registration, 1-800-334-8644.

April 4

37th Annual Clinical Conference (Obici Hospital), *Suffolk*. George J. Carroll, MD, 804-934-4000.

April 6-8

Mammography: Current Concepts and Interpretation (Eastern Virginia Medical School), *Williamsburg*. CME Office, 804-446-6140.

April 6-8

Annual Ophthalmology Conference: Cornea and External Disease (Medical College of Virginia/VCU), *Williamsburg*. CME Office, 804-786-0494.

April 12

Newer Infectious Diseases: Plagues of the 1990s (Southwest Virginia Medical Society), Ramada Inn, *Wytheville*. 4½ credit hrs. Kenneth J. Walker, MD, 703-921-3636.

April 19-21

Annual Meeting, Virginia Society of Otolaryngology/Head and Neck Surgery, *Alexandria*. Donna Scott, 804-353-2721.

April 19-22

10th Edition, Clinical Electrocardiography: Basic Concepts and Interpretation (Eastern Virginia Medical School), *New Orleans, Louisiana*. CME Office, 804-446-6140.

April 20-22

12th Annual Emergency Medicine for the Primary Care Physician (Medical College of Virginia/VCU), *Williamsburg*. CME Office, 804-786-0494.

April 21

Regular meeting of the Medical Society of Virginia's Council, *Richmond*. James L. Moore, Jr., 804-353-2721.

April 24

Common Problems in Primary Care (Fairfax Hospital), *Falls Church*. Cathy Sarraf, 703-698-2572.

April 26-29

11th Edition, Practical Dermatology for the Primary Care Physician (Eastern Virginia Medical School), *New Orleans, Louisiana*. CME Office, 804-446-6140.

April 27

2nd Annual Virginia Heart Center Symposium (Fairfax Hospital), *Falls Church*. Cathy Sarraf, 703-698-2572.

April 27-29

25th Annual Pediatric Springfest (Medical College of Virginia/VCU), *Williamsburg*. CME Office, 804-446-6140.

continued on page 132

You're Invited

The purpose of VaMPAC is to support political candidates who are sympathetic to physicians' concerns on medical and health issues by offering services, monetary support, and warm bodies.

VaMPAC is the non-partisan political action arm of the Medical Society of Virginia and is for neither Republicans nor Democrats but for our political friends.

A goal of VaMPAC is to convince those who are neutral, or even hostile, to our concerns to become our friends. And if we cannot make a friend, we try not to make an enemy, because some day we may be able to make him/her a friend.

Another goal of VaMPAC is to educate physicians about political issues, which sometimes is the more difficult task.

VaMPAC is run by a Board of Directors of thirty-five MSV members who desperately want and need your input. They invite you to come to their meetings to see how your political action committee functions. Here is the 1990 meeting schedule:

**April 17, 2:30 pm, Richmond,
place to be announced**

**June 16, 10:30 am, Richmond,
MSV headquarters**

**September 22, 10:30 am, Richmond,
MSV headquarters**

**November 1, the Homestead,
time to be announced**

If you have questions, call Jo Parkin
in the VaMPAC office at
MSV headquarters, 804-353-2721.

VaMPAC

MORE MEETINGS

May 3-6

Annual Meeting of the Virginia Chapter, American College of Radiology, the Homestead, Hot Springs. Patricia R. Berry, 703-669-8312.

May 4-6

10th Annual Clinical Concerns in Primary Care (Medical College of Virginia/VCU), Williamsburg. CME Office, 804-446-6140.

May 5

Nutritional Support of the Trauma Patient (Fairfax Hospital), Falls Church. Cathy Sarraf, 703-698-2572.

May 8

8th Annual Ethics Symposium: The Dilemma of Fetal Viability—Medical and Legal Impact on the Delivery of Health Care (Fairfax Hospital), Falls Church. Cathy Sarraf, 703-698-2572.

May 10-13

Annual Meeting, Virginia Orthopaedic Society, Wintergreen. Donna Scott, 804-353-2721.

May 14-18

Topics in Clinical Medicine (Johns Hopkins), Baltimore. 40 credit hrs. Fee, \$650. CME Office, 955-2959.

May 25-27

Advances in Pediatrics (American Academy of Pediatrics), Hilton Head Island, South Carolina. 16 credit hrs. CME Registration, 800-421-0589.

May 26-28

11th Annual Urogynecology and Pelvic Surgery (Medical College of Virginia/VCU), Williamsburg. CME Office, 804-786-0494.

June 4-8

6th Edition, Family Medicine Review Course (Eastern Virginia Medical School), Virginia Beach. CME Office, 804-446-6140.

June 4-8

General Surgery Seminar: Update and Review (Lloyd Noland Hospital), Hilton Head Island, South Carolina. CME Office, 205-783-5276.

June 6-10

7th Annual Meeting of the Southern Orthopaedic Association, Maui, Hawaii. Kathy McLendon, 205-945-1848.

June 7-9

Update in Obstetrics (American College of Obstetricians/Gynecologists), Norfolk. 16 credit hrs. Fee, \$515. ACOG Registrar, 800-673-8444.

June 7-10

14th Annual Postgraduate Course on Rehabilitation of the

Brain-Injured Adult and Child (Medical College of Virginia/VCU), Williamsburg. CME Office, 804-786-7290.

June 11-15

Adult Infectious Disease Seminar: Current Update (Lloyd Noland Hospital), Hilton Head Island, South Carolina. CME Office, 205-783-5276.

June 14-16

35th Annual Great Smokey Mountains Pediatric Seminar (University of Tennessee), Gatlinburg, Tennessee. Abby Hall, 615-544-9190.

June 15-16

Arrhythmias: Interpretation, Diagnosis and Management (Medical Education Resources), Williamsburg. Deborah Helton, 800-421-3756.

June 19-23

Pediatric Infectious Disease Seminar: Current Update (Lloyd Noland Hospital), Hilton Head Island, South Carolina. CME Office 205-783-5276.

June 21-24

Annual Meeting, Virginia Society of Ophthalmology, Norfolk. Donna Scott, 804-353-2721.

June 22-24

Clinical Pediatrics (American College of Pediatrics), Washington, DC. 16 credit hrs. CME Registration, 800-421-0589.

SERIAL LEARNING PROGRAMS

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June 17-29, Orthopedics; 60 credit hrs; tuition, \$1000

June 24-29, Family Practice, 26 hours, \$400

July 8-11, Addiction Medicine, 14 hrs, \$360

July 9-10, Child Abuse, 12 hrs, \$150

July 9-13, Allergy/Asthma/Dermatology, 18 hrs, \$400

July 9-13, Pediatrics, 16 hrs, \$400

July 15-17, Gastroenterology, 12 hrs, \$300

July 17-20, Urology, 15 hrs, \$375

July 17-20, Surgical Techniques, 15 hrs, \$400

July 22-26, Ophthalmology, 18 hrs, \$450

July 23-27, Anesthesiology, 15 hrs, \$375

July 29-August 3, Obstetrics/Gynecology, 22 hrs, \$500

July 30-August 2, Diabetes, 15 hrs, \$375

July 30-August 3, Otolaryngology, 18 hrs, \$450

August 12-16, Emergency Medicine, 24 hrs, \$400

August 12-16, Forensic Medicine, 34 hrs, \$400



Call For Papers

3rd Annual Cumberland Hospital Conference

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- Accepted authors are eligible for a reduced tuition of \$100 (full tuition is \$175). If more than one person is presenting the paper, all will receive the reduced tuition.
- All submissions must be postmarked before May 1, 1990. If selected, the author's name, title and topic will appear in the conference brochure. Authors will be notified by May 15, 1990.

Submission Requirements

- In a maximum of two typed pages, tell the conference committee about your proposed presentation and why attendees will be interested in the topic.
- Include with the two typed pages, your Curriculum Vitae.
- Also include with the proposal any other information which may assist the committee make a decision.
- All materials should be mailed to:
Conference Committee
Cumberland Hospital
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New Kent, VA 23124

Conference Location

The Conference will be held at the Williamsburg Hilton Hotel. The conference facility is located only a few miles from Colonial Williamsburg and at the entrance to "Busch Gardens, the Old Country."

Additional Information

For additional information on the conference, contact the Information Office of Cumberland Hospital for Children and Adolescents at 1-800-368-3472.

VIRGINIA MEDICAL CLASSIFIED

Virginia Medical classified ads accepted at the discretion of the Editor. Rates to Medical Society of Virginia members: \$15 per insertion up to 50 words, 25¢ each additional word. To non-members: \$30 per insertion up to 50 words, 25¢ each additional word. Deadline: 5th day of month prior to month of publication. Send to the Advertising Manager, 4205 Dover Road, Richmond VA 23221.

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WHO'S WHO

Dr. Anas M. El-Mahdi, chairman of the Department of Radiation Oncology and Biophysics at Eastern Virginia Medical School, is to be the recipient of the medical staff award for outstanding contributions to medicine at the Obici Hospital's 37th annual conference on April 4 in Suffolk. An Egyptian by birth, Dr. El-Mahdi earned his MD degree at Cairo University (1959) and a doctorate in science at Johns Hopkins University (1970). He is a fellow of the American College of Radiology, belongs to numerous national societies relevant to the sciences, and has published in excess of 175 cancer-related articles in medical journals in this country and abroad.

For his leadership in wildlife conservation in Virginia, a portrait of **Dr. William A. Pennington** has been given a place of honor in the Virginia Wildlife Federation's headquarters in Virginia Beach. The Buckingham County physician and former Virginia legislator helped organize the Buckingham Game Conservation Club in 1947; a year later, when the club became an arm of the National Wildlife Federation, he became the statewide organization's first president. Today the VWF has a membership of 16,000, and Dr. Pennington is on its board of directors.

Fellowship in the American College of Surgeons was conferred on **Dr. William E. Broach III**, Danville, at the College's 1989 clinical congress.

And a Danville nephrologist, **Dr. Michael A. Moore**, has been elected to a fellowship in the Council on Hypertension Research for the American Heart Association. As a representative of the AHA, Dr. Moore chairs the National Institute of Health's High Blood Pressure Education Coordinating Committee.

For "outstanding contributions, both to the professional world and to the community as a whole," **Dr. Susan J. Mellette**, Richmond, was named 1989 Professional of the Year by the Virginia Association of Professions. Dr. Mellette is professor of oncology at the Medical College of Virginia and head of the school's cancer rehabilitation program.

Dr. H. Joseph Williams, family physician in Staunton, was one of five persons designated winners of the 1989 Distinguished Service Awards of the National Interscholastic Athletic Administrators Association. The awards were presented in December at a meeting of the

NIAAA in Dallas, where Dr. Williams was praised for his 40 years as team physician for the football and basketball teams of Staunton's Robert E. Lee High School. The 1989-90 basketball season will be his swan song on the athletic field, Dr. Williams says, although at 72 he's still up for private practice.

Two Medical Society of Virginia members received distinguished faculty awards from Virginia Commonwealth University during the university's annual convocation last month. They are **Dr. Leo J. Dunn**, chairman of the department of obstetrics and gynecology at the Medical College of Virginia, who was given the Distinguished Service Award, and **Dr. Hyung Mo Lee**, MCV's chairman of vascular and transplantation surgery, who received the University Award for Excellence.



Dr. Russell D. Evett, MSV councilor for the 2nd district, went back to med school recently as the Medical Society of Virginia's emissary to the scholarship recognition ceremony at Eastern Virginia Medical School. He is shown above, flanked by the two EVMS students who shared the Society's scholarship. They are Maria Tabora (M-92), left, and Jacqueline Henderson (M-92). Dr. Evett's alma mater, the Medical College of Virginia/VCU, also received MSV scholarship money, and so did the University of Virginia School of Medicine.

Photo courtesy Eastern Virginia Medical School

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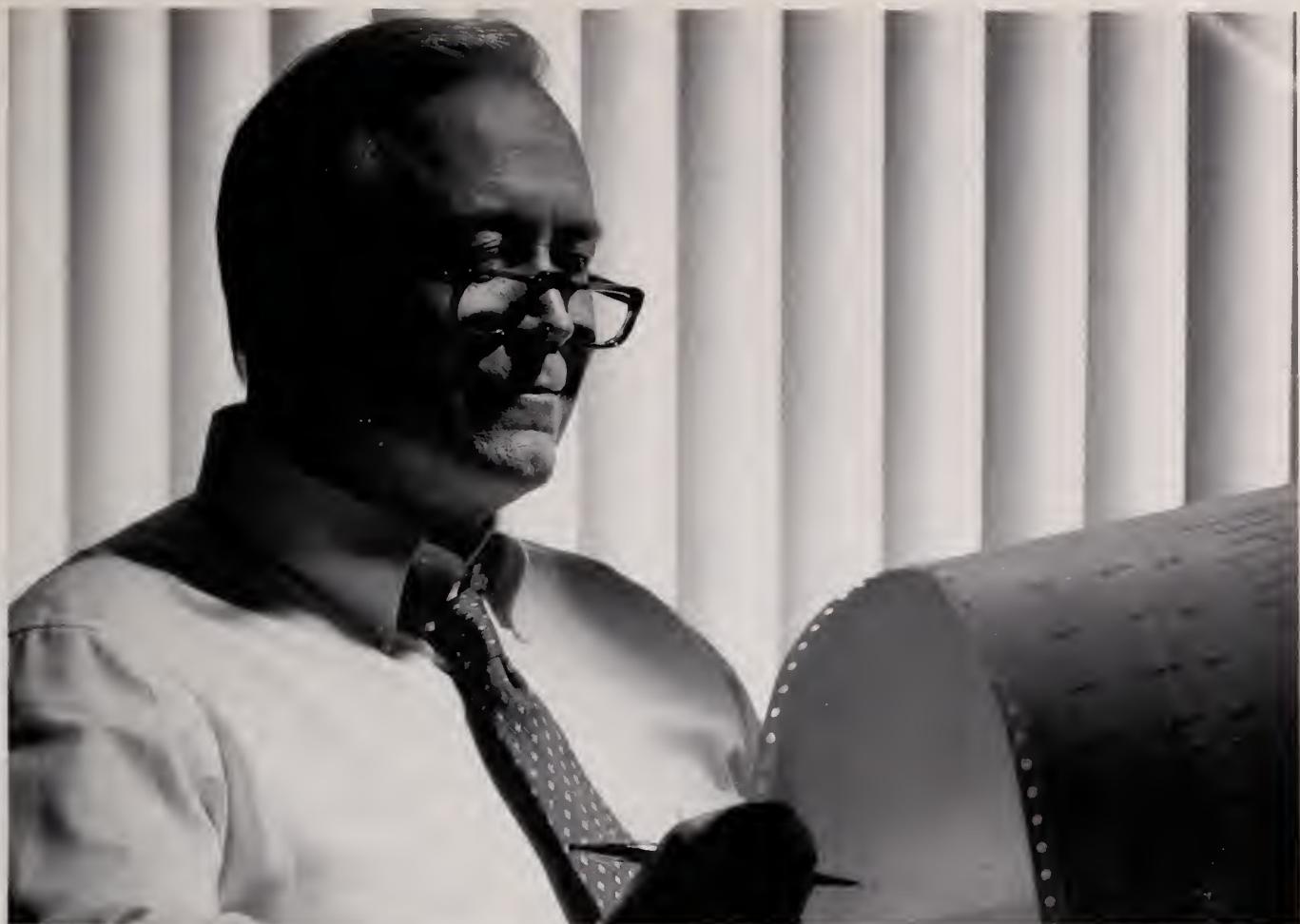
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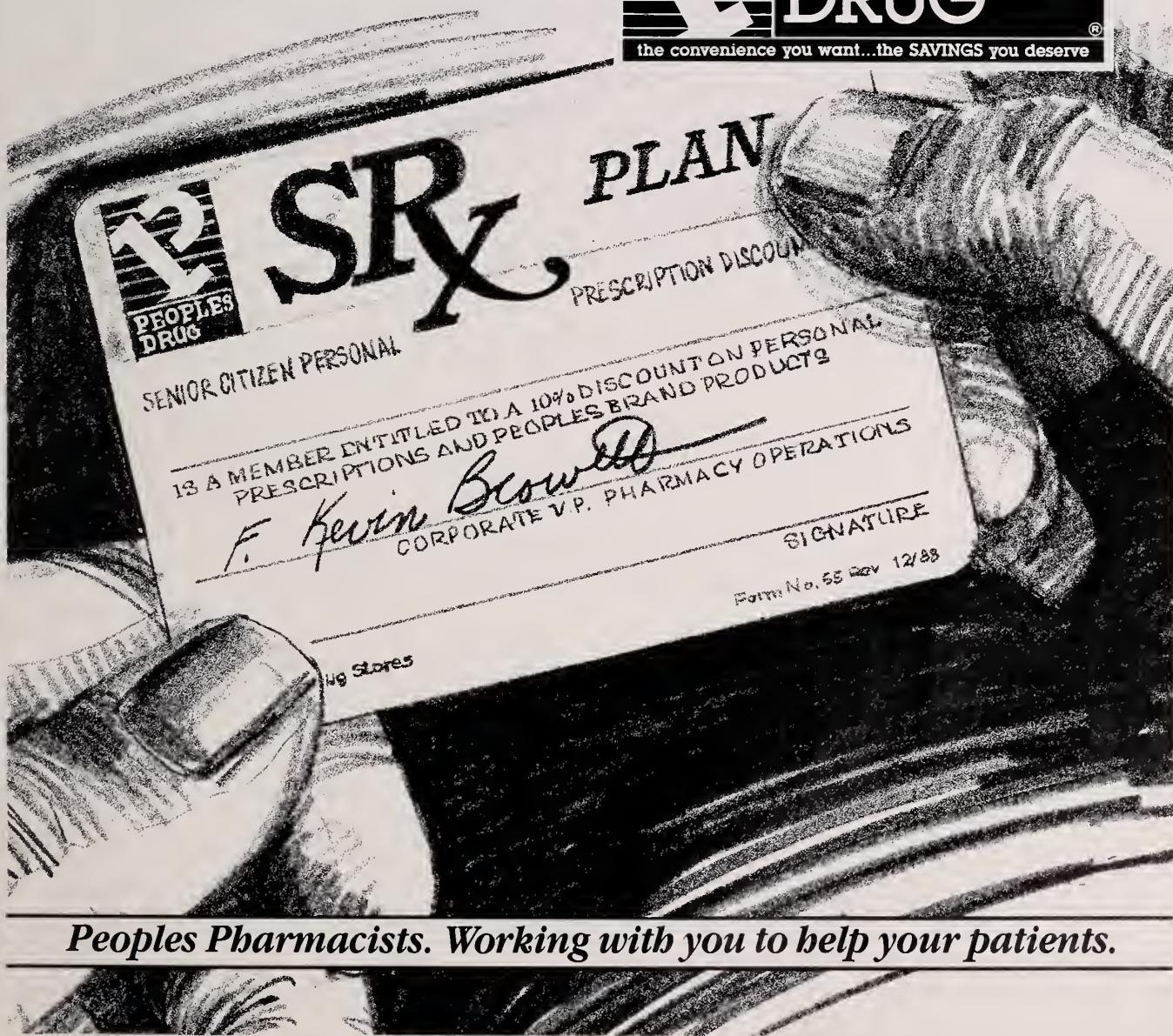
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LETTERS

Automated CBC count may obscure L-tryptophan toxicity diagnosis

L-tryptophan toxicity is now an established syndrome. Here is a summary case report to add to that of Dr. Becker et al.¹

A 58-year-old white female was seen initially in the office on November 10, 1989, cyanotic, dyspneic and appeared near death. Chest x-ray was compatible with acute respiratory distress syndrome. She was immediately hospitalized and initial blood gases had astonishing findings of pH of 7.49; pCO₂, 31; pO₂, 27; with initial blood count of 15,400, segments 80%, bands 4% and lymphs 10%. No eosinophils were reported. The patient had to be intubated. The dramatic report of L-tryptophan toxicity appeared in the local newspaper that weekend and because of this the lab work was reviewed. The error on my part was forgetting that in automated counting of a CBC, eosinophils are not counted. Knowing the patient had peculiar eating habits (once she asked me to give her weight-reducing pills when she weighed only 85 lbs), I questioned her on the respirator and learned she was taking 10 grams of L-tryptophan a day. I then requested a total eosinophil count and a differential count done by hand. This confirmed the diagnosis. The total eosinophil count was 1,513 and a WBC of 14,500, normal count being 400. With this information the patient was started on large doses of IV steroids, with remarkable clearing of a puzzling lung diagnosis, and she was quickly restored to normal health.

Because of automated counting it is probable that many cases of L-tryptophan toxicity have been missed.

Milton Ende, MD

121 South Market Street
Petersburg VA 23803

1. Becker DM, Habib-Hong S, Smith SM. Eosinophilia and myalgia associated with L-tryptophan use: case report. Va Med 1990;117:68-70

Dr. Becker comments: Dr. Ende makes an important point. Most hospitals rely on automated CBC analyzers that can only estimate the WBC differential by noting variations in WBC size; thus in most hospitals an accurate WBC differential must be done manually, usually with special stains, such as peroxidase. However, there are machines that can automate the staining process and count thousands of WBCs in order to provide an accurate WBC differential from which an eosinophil count can be reliably estimated. In any

case, an eosinophil count is indicated for all patients with unexplained myalgia, myopathy, neuropathy, or pneumonitis, and knowledge of L-tryptophan use should lead directly to this test.

Help available to doctors/parents involved in parental placement adoptions

The 1989 General Assembly passed changes in the parental placement adoption laws. These changes were intended to protect the interests of all parties in parental placement adoptions. One of the new requirements in the law is that birth mothers be offered counseling about the adoption decision and other options. In addition, home studies are required to be performed by licensed child placement agencies, either public or private. Public agencies have indicated that they may not be able to perform these studies because of their present case loads.

Children's Home Society of Virginia has been serving the needs of birth mothers, children and adoptive families for 90 years. The Society, a non-profit, non-sectarian agency, has professional staff to respond to the rising demand for these services throughout the state and is prepared to work with all interested parties for better understanding of these new requirements. In addition, Children's Home Society will work with local groups to conduct workshops on the process of adoption.

Should physicians have any questions regarding the new requirements for parental placement, they should call Mr. Peter Pufki, Executive Director, or Ms. Lois Gibbs, Child Placement Supervisor, at Children's Home Society of Virginia, 804-353-0191.

Grayson A. Harding

President,
Children's Home Society of Virginia
4200 Fitzhugh Avenue, Richmond VA 23230

In praise of John Lynch, “inspiring and dedicated leader”

On the morning of July 16, 1989, in the presence of his family at St. Luke's Hospital, John Phillip Lynch “fell on sleep and was gathered unto his fathers.” The aphorism that “life may be and was meant to be an immortal youth” seemed well nigh to reach its complete fulfillment in the life history of Dr. Lynch.

Over 100 physicians associated with the McGuire Clinic formed the avant garde of medicine in Richmond. At their forefront John Lynch served as an inspiring and dedicated leader in the advancement of health sciences and especially medicine and nursing. While the practice of medicine has been radically affected by the government asserting its influence, Dr. Lynch and his associates held unwaveringly to the

traditional relationship between patient and physician and maintenance of the highest standards of professionalism in resistance to bureaucracy and the rigidity of standardization.

Dr. Lynch's record of community service was exemplary. In the 1950s he served the Richmond Urban League as a member of the board during its trying time following the transition of Richmond from a rural community to an industrial and urban center. He was heavily involved in organization and legislation governing the nursing profession and became consultant to the Governor's Council on Nursing in 1968-1969. To the St. Luke's Hospital School of Nursing, which has produced nearly 1,000 alumnae, John Lynch contributed unstintingly his tutelage, inspiration, and direction. A pioneer in the quest for quality of care in nursing homes, rehabilitation of the disabled, and legislation favoring older workers, he contributed in an advisory capacity to a number of Richmond hospitals, nursing homes, and rehabilitation centers, lending the

knowledge and expertise of nearly 50 years of active and widely acclaimed involvement in medicine. He was the first chairman of the Gerontology Committee of the Richmond Area Community Council and chairman of the Committee on Aging of the Medical Society of Virginia from 1979 to 1984. He was a committed member of the First Presbyterian Church, serving as an elder, and was cochairman of the Committee on Christianity and Health, Hanover Presbytery, Presbyterian Church of the United States.

Dr. Lynch's philosophy, ideas, and ideals have permeated our society, and so he will continue to be with us and live on in our minds and our hearts.

Walter W. Regirer

President, Health of Virginia
2420 Pemberton Road
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Editors' Note: For another tribute to Dr. Lynch, see the Obituary columns on page 169.



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Waste Disposal: How to Handle the New Regulations

For many physicians, the new regs will add up to a contract with a waste handler. True, the regulations provide for do-it-yourselfers, as this article sets forth, but this route is not applicable to all practices.

POISED to go into effect on Tuesday, May 2, are Virginia's new Infectious Waste Management Regulations, which define infectious waste and stipulate specific requirements for its collection, transport, treatment and disposal. Thus is the State of Virginia responding to the recent hullabaloo when medical items washed up on New Jersey beaches. Congress rushed to pass the Medical Tracking Act; the states are following up with regulations. It makes no matter that those New Jersey beach items were never proven to be infectious. What matters is that the public *perceives* such waste as infectious. That perception is what state officials are trying to grapple with.

What Virginia's new regulations give physicians to grapple with are a long list of items defined as infectious and rigorous packaging/treatment/disposal specifications for that waste. For many physicians, this will add up to a contract with a commercial waste handler and a corresponding hike in office overhead. True, the regulations provide for do-it-yourselfers, as this article sets forth, but this route takes some doing and is otherwise not applicable to all practices.

In any case, it is important to know exactly what the regulations say and mean so that you can evaluate intelligently the solicitations of waste handlers and comply with the regulations in the most cost-effective manner.

THE WASTE

The regulations stipulate that waste defined as infectious (Table 1) must be handled as follows:

- 1) Infectious waste cannot be placed in a landfill without undergoing treatment to render it non-infectious.
- 2) The stipulated treatment is either incineration or steam sterilization.

See related editorial, page 166.

No other treatment will be considered acceptable except by special approval of the director of Virginia's Department of Waste Management.

It should be noted that the regulations exclude certain items from the infectious designation. They appear in Table 2.

THE GENERATOR

The regulations apply to "all persons who generate infectious wastes; own or operate infectious waste management facilities or allow

This article and accompanying information were developed jointly by John H. Keene, DrPH, and Ann Gray. Dr. Keene is president of Biohaztec Associates, a biosafety consulting firm at 924 Castle Hollow Road, Midlothian VA 23113. Mrs. Gray is executive editor of Virginia Medical.

As a service to Medical Society of Virginia members, most of the information published here was preprinted in newsletter format and mailed early last month.

infectious waste management facilities to be operated on their property in this Commonwealth . . . except those specifically exempted or excluded elsewhere in these Regulations."

Since physicians generate wastes defined by the regulations as infectious, they are subject to compliance. As the generator, the physician is responsible for the appropriate packaging and labeling of that waste and cannot delegate this task to a contractor or other agent.

From other requirements involving transport and treatment, however, the physician-generator may qualify for money-saving exemptions, as follows.

EXEMPTIONS

According to Section 3.1.E, the regulations do not apply to health care professionals who generate waste defined as infectious in the provision of services in their own offices or in the private homes of patients, provided the waste is disposed of as specified:

1. With respect to defined infectious waste other than sharps, if the practitioner accumulates no more than 64 gallons of waste, packages it as required (see Packaging/Labeling below), and delivers it within 14 days to a permitted infectious waste storage or treatment facility, as defined by Section 3.1.E.3, that practitioner is exempted from all other sections of the regulations.

2. With respect to sharps, if the sharps are packaged in rigid, leak-proof and puncture-resistant containers, are labeled as required, and are transported to a permitted waste treatment or storage facility, the practitioner is exempted from all other requirements of the regulations.

3. With respect to transporting either or both of the above, if the physician or one of his/her employees transports the packaged/labeled waste to a treatment or storage facility, the material becomes exempt from Section 4.2.B.4, which requires that the material be additionally contained in a rigid, double-walled, corrugated fiberboard box.

If, however, the physician hires a waste hauler to do the transporting, the boxing requirements of Section 4.2.B.4 apply, with the attendant labor and costs.

CONTROLLING COSTS

What else can physicians do to minimize the economic impact of these new regulations? Here are some possibilities.

Auditing the Waste

Wastes not defined as infectious, i.e., "normal" wastes, need not be treated and may be disposed of in the conventional way. Accordingly, this non-infectious waste should be carefully segregated so that it does not add to the burden and cost of disposing of the infectious wastes.

For the removal, transport, treatment, and disposal of infectious material, the waste handler will charge by the pound and, because of the presumed hazard level, at a premium rate. Accordingly, the generation of this waste should be kept at a minimum.

A review of Tables 1 and 2 will identify the quantity and types of wastes being generated within a time period, so that an infectious waste control plan for the practice can be developed.

Segregating the Waste

Throughout the office, provide two containers for waste disposal,

Table 1. Waste Defined by the Regulations as Infectious and Subject to Control.

1. Cultures and stocks of microorganisms and biologicals. Discarded cultures, stocks, specimens, vaccines and associated items likely to have been contaminated by them.
2. Blood and blood products (free-flowing blood).
3. Pathological wastes.
4. Sharps—hypodermic needles, syringes, scalpel blades, Pasteur pipettes, broken glass, etc.
5. Animal carcasses, body parts, bedding and related wastes when animals have been deliberately infected with organisms likely to cause disease in healthy humans.
6. Residues or contaminated soil, water or other debris resulting from the cleanup of a spill of infectious waste.
7. Any waste contaminated by or mixed with infectious waste.

Table 2. Items Excluded from the Regulations.

1. Used products for personal hygiene (i.e., diapers, facial tissues and sanitary napkins).
2. Materials, not including sharps, containing small amounts of blood or body fluids but no free-flowing or unabsorbed liquid.

one for regulated waste, the other for non-regulated, or normal, refuse. All office personnel must be thoroughly informed as to the identification of regulated waste and the method for segregating it.

Packaging/Labeling the Waste

As noted above, the regulations make perfectly clear that the physician-generator of infectious wastes is responsible for the appropriate packaging and labeling of that waste; the responsibility for any portion of the procedure cannot be delegated to a contractor or other agent who is providing services to the generator. This applies whether or not the physician-generator is exempt from other parts of the regulations.

Further, the packaging and labeling must be performed in compliance with Section 4.2 of the regulations as follows:

1. All liquids must be contained in sturdy leakproof containers that resist breaking.
2. Sharps must be collected at the point of generation in puncture-resistant containers.
3. The above waste and all other regulated infectious waste (Table 1) must be contained either
 - in two leak-proof plastic bags (i.e., double-bagged), each bag capable of passing the ASTM 125-lb drop-weight test and each sealed separately, or

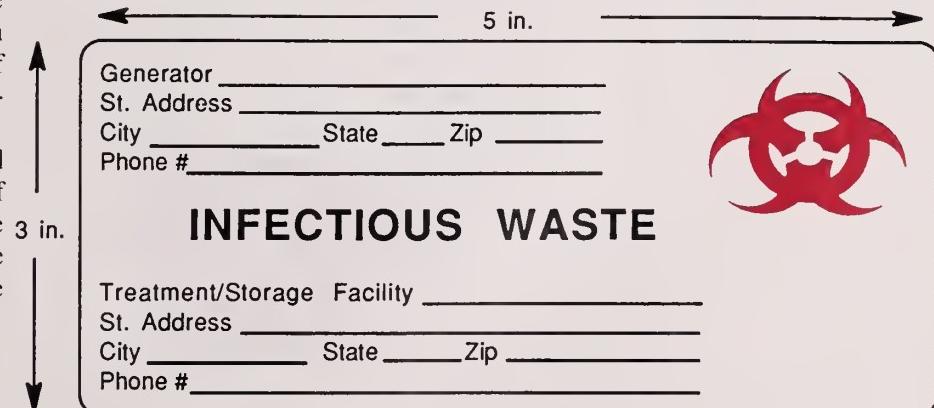


Fig. 1. Infectious waste label. Biohazard symbol must be printed in red.

Enforcement: 'No Knocking on MDs Doors'

Regulations by a state agency empowered by Virginia's General Assembly have the force of law, and the new Infectious Waste Regulations fit that category. Thus any intentional violation can be prosecuted as a felony, and civil penalties can also be assessed, according to Robert Wickline, environmental engineer with the Department of Waste Management, who has been designated to head up implementation of the new regs.

Asked about policing mechanisms, Wickline said he had planned to add a couple of persons to the department's enforcement staff, but the current budgetary crunch had wiped out that idea and he will borrow staff from the solid waste area. In any event, he emphasized, "There won't be anybody going around knocking on doctors' doors or anything like that." Instead, enforcement will focus on the waste handlers' operations.

Wickline is aware that doctors are bearing an increasing burden of regulations. He feels that physicians in urban areas will be able to adjust fairly readily to the new infectious waste strictures, but he expressed concern about physicians in rural areas, where waste handlers may impose higher prices for collection. "We haven't figured out a way to help them yet, but we're trying," Wickline said. Meantime, he is in hope that local hospitals will provide some assistance.

The Governor's Waste Management Team

Director of the Virginia Department of Waste Management is Cynthia V. Bailey, who was appointed by former Gov. Gerald L. Baliles and reappointed by Gov. L. Douglas Wilder. A graduate of the University of Richmond School of Law, Director Bailey was an assistant attorney general during Baliles' term as Virginia's attorney general.

The seven members of the Department's Waste Management Board are also appointed by the governor. They are selected for their expertise in areas pertinent to environmental issues. The current members are James R. Craig, PhD, Blacksburg, chairman; James A. Davis, PhD, Winchester; Edward I. Long, Lawrenceville; Michael Markels, Jr., PhD, Springfield; Joan MacCallum, Lynchburg; Frank H. Miller, Jr., Hampton; and Heather Wicke, PhD, Arlington.

- in one leakproof plastic bag inside a double-walled corrugated fiberboard box or equivalent rigid container meeting Department of Transportation standards.

4. For the waste that is to be steam sterilized, the plastic bags must be orange in color. They must be marked with autoclave tape. Waste destined for incineration must be placed in bags that are red in color.

5. All bags must be sealed against all possibility of leakage by lapping the gathered open end and binding with tape or a closing device.

6. If the waste is to be transported to a storage or treatment facility by a waste hauler, the plastic bags must be enclosed in a double-walled, corrugated fiberboard box as described above.

7. Figure 1 shows a sample label. Note that it is 3×5 inches in size, that the name/address/phone number of both the generator and the person or facility to whom control of the waste is being transferred are given, that the words "infectious waste" are in large, bold type, and the universal symbol for biohazards appears on the label in red. All this must be printed in indelible ink.

It is up to the physician to acquire all the packaging/labeling supplies specified above. A medical supply house may be the resource for sharps containers. The boxes may be procured from a waste handler. As for the label, you will probably want to have these printed especially for your use.

Treating the Waste

The regulations require steam sterilization of regulated infectious waste for a minimum of 90 minutes at 221° F at 15 pounds pressure. Thorough testing must be performed on the sterilizer and documentation kept for each load of waste sterilized. Documentation must include 1) date, time and operator of each run; 2) type and approximate amount of waste treated; 3) post-treatment reading of temperature-sensitive tape; 4) dates and results of calibration of the sterilizer; and 5) results of



spore treating performed for the month in which sterilization is done.

In the unlikely event the physician has access to an autoclave of sufficient capacity and treats the bags of waste as specified, then the steam-treated waste is considered no longer infectious and may be disposed of as normal trash. The waste must be packaged in orange plastic bags as cited above, however, and the autoclave tape must indicate that the material has been sterilized. In addition, the physician-generator must have complete documentation on file for each treatment run.

Transporting the Waste

If, as is likely, the practitioner cannot treat the regulated infectious waste on-site, it must be transported to a storage or treatment facility. You will remember the first listed exemption above, allowing for the private transport of infectious waste by the physician or the physician's employees provided that 1) the volume of waste is less than 64 gallons; 2) it has been stored for no more than 14 days; and 3) it is properly packaged in plastic bags and duly labeled.

If, however, the waste is taken to a storage facility, the operator of that facility will have to box it for the trip to a treatment facility, and there will no doubt be some charge for this service.

Training the Staff

All personnel in the doctor's office must be informed as to the specific methods for identifying, segregating, treating (if it is done on-site), and transporting of infectious waste. Those involved in these procedures should understand thoroughly the potential hazards involved and be informed as to the appropriate methods for protecting themselves from exposure. It is also important to document that the employees have received the training and to update that training periodically.

Protecting the Plan

"If anything can go wrong, it will." It is important to have alterna-

The Cost: It's Worse in New Jersey

In his editorial on page 166, Dr. Raymond S. Brown chronicles the increased costs his practice has incurred in order to meet increasingly rigorous waste disposal mechanisms, specifically his group's higher waste handling tab. In New Jersey, doctors not only have the treatment/handling costs to contend with but are also subject to an annual fee laid on by the State Department of Environmental Protection. Originally, when New Jersey's new regulations were passed early in 1989, the fee was set at \$528 for every waste generator in the state—including every physician-generator, but lobbyists for the Medical Society of New Jersey moved in on the legislature, and the following schedule of maximum charges was substituted: Under 50 lbs/yr, \$100; 50-200 lbs/yr, \$300; over 200-300 lbs/yr, \$500; over 300-1000 lbs/yr, \$1000; over 1000 lbs/yr, \$3500.

Who's Who in Waste Handling in Virginia

Three waste handlers account for most of the medical waste disposal in Virginia: Browning Ferris Industries, Waste Management, Inc., and Incendere, Inc. BFI and WMI are huge national firms, veterans of the waste disposal business. Incendere is a new firm headquartered in Norfolk, the only one of the three with its own treatment facility in Virginia.

- BFI's headquarters are in Houston, Texas. It has nine operating locations in Virginia, and its familiar blue trucks with the BFI logo will go anywhere in the state to collect medical waste, its representatives say. For details, call the Richmond office at 804-222-7070.

- WMI, which is headquartered in Oak Brook, Illinois, previously operated only in Northern Virginia (Alexandria, Arlington, Fairfax Counties) but is moving further into the state by setting up offices in Richmond, Norfolk, Newport News and Petersburg. For details, call the regional office in Charlotte, North Carolina: 704-364-1039.

- Incendere was founded in 1986 in Norfolk by two enterprising Virginians, the brothers Elliott and Dwight Shaubach. This is a regional operation, with additional offices in Richmond, Philadelphia, and Lexington, South Carolina. Its Norfolk treatment facility is comprised of three 25-ton incinerators. For details, call 1-800-USA-BURN.

tive plans for the transport and treatment of regulated waste in case something goes wrong. A hospital incinerator may no longer be available. What will you do? The employee assigned to take your waste to the storage facility gets sick and can't make the trip. Who will take it? Once your primary plan is developed, give it a long hard look and determine what problem areas might develop, then decide how you will handle the situation if something goes wrong.

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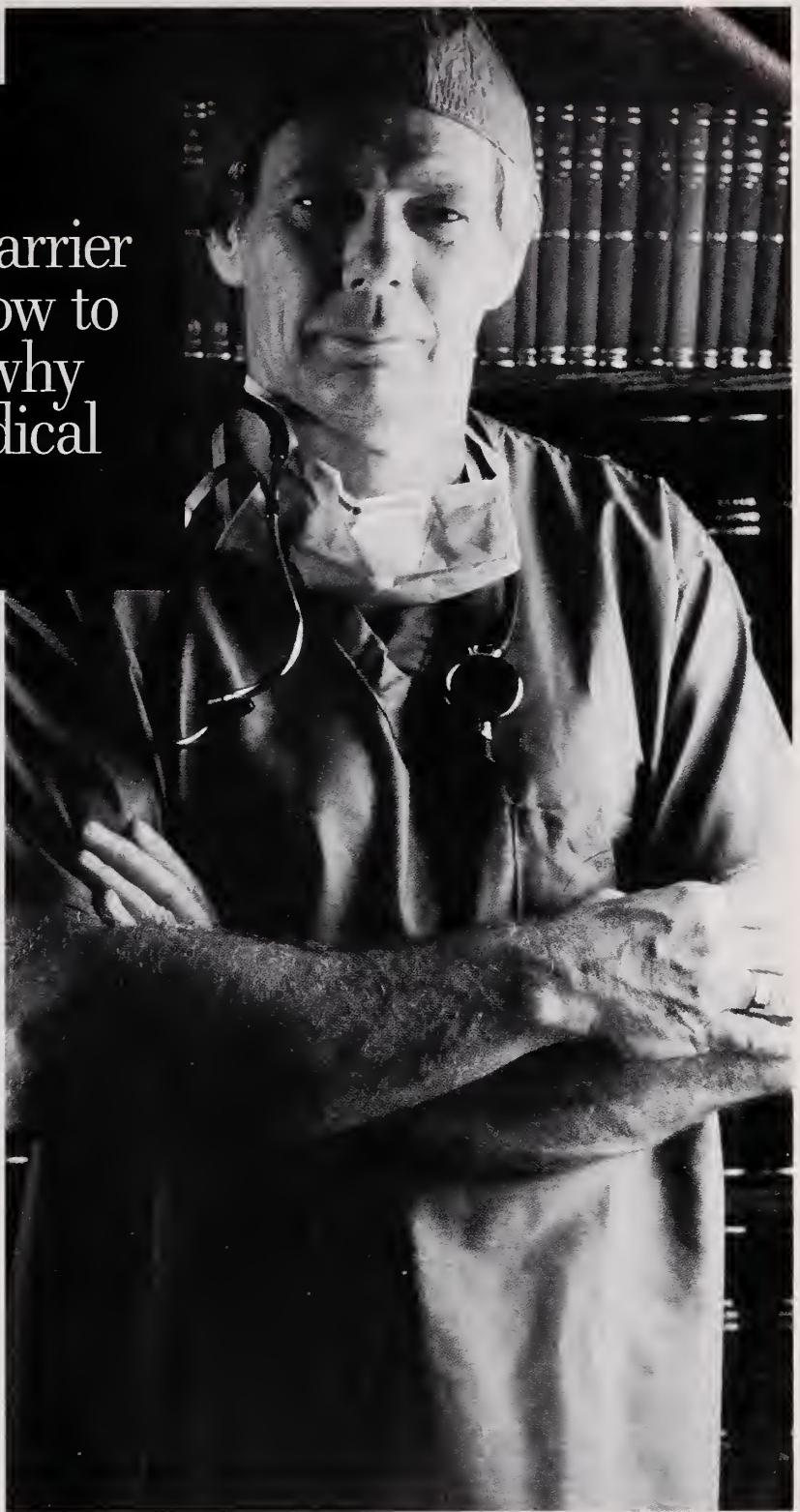
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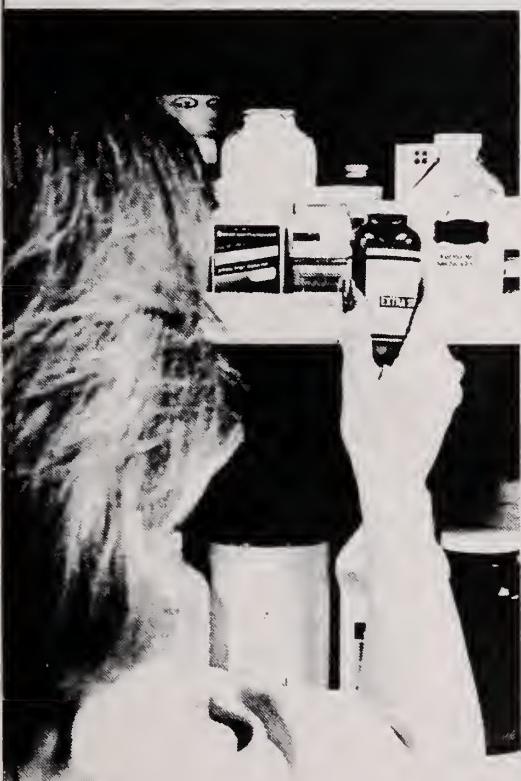


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House Dust Mites in Williamsburg, Virginia

Mark T. Lassiter, MA, and Norman J. Fashing, PhD,
Williamsburg, Virginia

House dust allergy is a common medical ailment. It has been well established that mites of the genus *Dermatophagoides* (house dust mites) are an important source of allergens and that mite counts greater than 300 per gram of dust are associated with symptoms of asthma. A survey of 22 houses in Williamsburg, Virginia, during the month of August revealed that all had mite populations exceeding this number. This may explain in part the high incidence of allergy in the Williamsburg area.

ALLERGIC REACTIONS to house dust in the form of perennial rhinitis, asthma, atopic dermatitis, and possibly urticaria are a common medical ailment throughout much of the world.¹ Mites, especially *Dermatophagoides farinae* and *D pteronyssinus*, play a major role in the production of dust allergens.^{1,2} If mite-associated dust allergens are to be eliminated from houses and work places, it is important to know the species composition, densities and locations of mite populations. Surveys conducted in numerous locales worldwide have revealed that such parameters vary widely with geographic location and climatic factors, making it evident that such surveys must be undertaken on a "by-locality" basis.

The present study investigated mite populations inhabiting house dust in Williamsburg, Virginia, an

area with an extremely high incidence of allergy (Dr. Scott Pharr Jr., MD, otolaryngologist, personal communication). Objectives were 1) to determine the species of mites represented as well as their densities, 2) to determine whether population numbers differ in beds, sofas and rugs, and 3) to attempt to find associations between population densities and cleaning habits as well as other environmental conditions within houses.

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When this work was done, Mr. Lassiter was a graduate student at William and Mary. He is now in the Department of Entomology at North Carolina State University, Raleigh.

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Materials and Methods

Dust samples were collected from 22 middle-income, single-family dwellings in the Williamsburg area in 1982. Dust mite populations vary with the seasons and peak during late summer, typically August.²⁻⁷ All samples were therefore collected in August to provide maximal population estimates.

Samples were collected from four sites within each home: bed mattress, bed pillow, carpet beside bed, and sofa (usually in a different room). The collection apparatus consisted of a plastic vial 1 1/4 inches in diameter and 4 inches in length. A dispenser nozzle was inserted on one end of the vial to concentrate the vacuum, and a 44- μ m mesh screen was attached to the other to trap dust and mites. The mesh end of the vial was connected to the hose of a General Electric model PIC-200 vac-

uum cleaner. At each collection site, the nozzle (6.4-mm diameter opening) was passed over the substrate for one minute, vacuuming an approximately 116 cm² collection area as follows: 1) mattress—184 cm of the top seam including a corner at both the head and foot; 2) pillow—184 cm around the seam; 3) carpet—92 cm straight line beside the bed (the 92 cm distance was used to approximate the increased surface area of collecting both from the side of piling and the pile mat); 4) sofa—184 cm around the cushion seams. Each vial was weighed before and after the collection to determine the sample weight.

Shortly after collection, each sample was flushed from the collection vial into a 44-μm mesh sieve, using distilled water. The sieve containing the sample was stored in a sealed jar with a thin layer of 85% ethyl alcohol and analyzed at a later date.

For analysis, the sieve was removed from the storage jar and flushed with distilled water to remove the sample into a petri dish. Alcohol remaining in the jar was poured through the sieve and the sieve then checked for any remaining mites under a dissecting microscope. These mites were added to the sample. The petri dish containing the dust sample was then placed on a black surfaced grid and examined under a dissecting microscope. Small probes were used to break up consolidated debris and clusters of mites. All non-pyroglyphid species were separated out and mounted on slides for identification under a phase contrast microscope. The total number of pyroglyphid mites was then counted. To determine the number of each species of *Dermatophagoides* present in a given sample, a species ratio was utilized. To establish the ratio, a random sample of 50 adults was separated out, slide mounted and identified under a phase contrast microscope. If there were less than 50 adults in the sample, all were examined.

A questionnaire designed to obtain information concerning environmental conditions within each house



Fig. 1. Male house dust mite. Bar = 200 micrometers.

was completed by the person responsible for home cleaning immediately following the collection of samples. Data collected concerned the age of the house, mattress and sofa; type of heating and cooling; type of carpet; type of bed covering; frequency of usage of bed and sofa; cleaning frequency for mattress, mattress cover, sheets, sofa and carpet; and presence of pets.

Results

Mites Represented. Although a given sampling site within a house was sometimes devoid of mites, mites were found in each of the 22 houses sampled. Table 1 provides a breakdown of the 9,387 total mites found in

the dust samples. Four suborders were represented—Astigmata, Oribatei, Prostigmata and Mesostigmata. Members of the genus *Dermatophagoides* accounted for 98% of all mites collected. *Dermatophagoides farinae* was present in all houses and made up 70% of the mites collected, whereas *D pteronyssinus* occurred in 21 of the 22 houses and made up 28%. Other astigmatid mites, primarily *Tyrophagus*, were present in nine houses but only in small numbers. Prostigmatid mites accounted for 1% of the total and were found in 16 houses. All but one were in the family Cheyletidae. Mesostigmatid mites were found in ten houses for a total count of 22 individuals. Oribatid mites were found in only five

Table 1. Mites Collected From House Dust in Williamsburg, Virginia.

Taxa Represented	Number of Mites Represented		Number of Houses	
	Total	Percent	Total	Percent
Astigmata	9219	98.21	22	100.00
Acaridae	23	0.24	9	40.91
<i>Tyrophagus</i> sp	22	0.23	9	40.91
Unidentified sp	1	0.01	1	4.55
Pyroglyphidae	9196	97.96	22	100.00
<i>Dermatophagoides farinae</i>	6547	69.74	22	100.00
<i>Dermatophagoides pteronyssinus</i>	2649	28.22	21	95.45
Oribatei	46	0.49	5	22.73
Prostigmata	100	1.06	16	72.72
Cheyletidae	99	1.05	16	72.72
<i>Acaropsis</i> sp	12	0.13	2	9.09
<i>Cheyletus</i> sp	20	0.21	5	22.73
Unidentified sp	67	0.71	14	63.64
Mesostigmata	22	0.23	10	45.45
Digamasellidae	1	0.01	1	4.55
<i>Digamasellus</i> sp	1	0.01	1	4.55
Dermanyssidae	1	0.01	1	4.55
<i>Dermanyssus gallinae</i>	1	0.01	1	4.55
Laelapidae	1	0.01	1	4.55
<i>Hypoaspis</i> sp	1	0.01	1	4.55

homes—one bed sample contained 40, whereas only six individuals were found in the other four houses combined.

Both *D. farinae* and *D. pteronyssinus* were found in 95% of the houses, with *D. farinae* dominant in 16 (76%) and *D. pteronyssinus* dominant in only five (24%) (Fig. 2).

Habitat Comparisons. Mites were found to inhabit mattresses and carpets in all 22 houses sampled, sofas in 21 houses, and pillows in 20 houses. The largest population occurred in the sofa in 50% of the houses, whereas in 45% of the houses the largest population was found in the mattress. In only one house was the largest population found in the carpet.

Mite densities in the different collection sites were compared in two ways: 1) actual number of mites observed and 2) the number of mites present per gram of dust (Table 2). Both sets of data—mite number per sample and mite number per gram of dust—were subjected to a one-way analysis of variance (ANOVA) followed by a Student-Newmann-Keuls test. Since data were in the form of counts, a square root transformation was used to provide a better fit to the ANOVA model.⁸ In both analyses, mite populations in sofas were not

significantly different in number from those in mattresses; however, mite densities in carpets and pillows were found to be significantly different from each other as well as from those in both the mattresses and sofas.

Effects of Environmental Conditions. No significant correlation was found between mite density and years in residence, house age, mattress age or sofa age. One-way ANOVAs were used to test whether mite densities were affected by environmental conditions. There was no significant difference in mite densities per gram of dust in regard to type of heat, bed age (≤ 10 yrs vs > 10 yrs),

bed use (≤ 8 hrs/day vs > 8 hrs/day), sheet change (1/wk vs < 1 /wk), mattress cleaning (yes vs no), carpet type (tight pile vs shag), carpet cleaning (≥ 1 /wk vs < 1 /wk), sofa age (≤ 5 yrs vs > 5 yrs), sofa cleaning (yes vs no), and presence of pets (yes vs no). A significant difference was found, however, in regard to sofa use. Those used less than five hours per day had significantly lower mite densities than those used five hours or more per day ($p = .01$). Since almost all homes were air conditioned and since most mattress coverings were cleaned, these variables were not analyzed.

Discussion

It has been well established that the most common mite species found in house dust are members of the family Pyroglyphidae, and the present study supports this fact (Table 1). Non-pyroglyphid species are generally accidentals in the house dust system. An exception to this are some species of the family Cheyletidae, which prey on *Dermatophagoides* and are therefore an important component of the house dust community.^{9,10}

We found only two representatives of the Pyroglyphidae, *D. farinae* and *D. pteronyssinus*, in Williamsburg dust samples. Other species, especially *Euroglyphus maynei*, have been recorded from homes in the U.S., although usually in small numbers.¹⁰⁻¹³ It is possible that other species were present in some of our

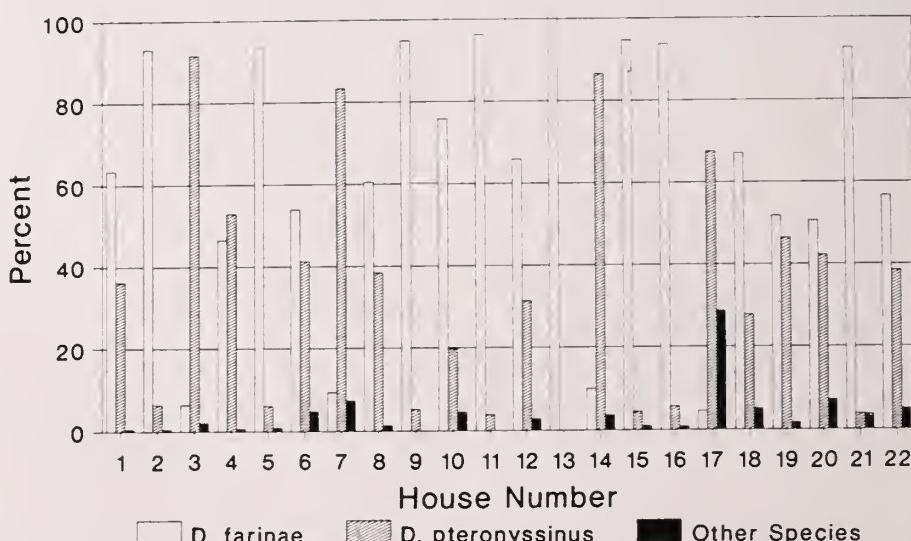


Fig. 2. Breakdown of mite species present in the 22 houses surveyed in Williamsburg.

Table 2. Summary of Mite Densities in Collection Sites in 22 Homes in Williamsburg.

Collection Site	Mean	Standard Error	Range
Total Mites Collected per Sample			
Mattress	132	2.63	7-988
Pillow	6	0.09	0-40
Carpet	59	0.87	5-341
Sofa	125	1.49	0-613
Mites Collected per Gram of Dust			
Mattress	4099	52.23	250-14500
Pillow	471	9.96	0-4000
Carpet	1483	30.91	73-9700
Sofa	4580	38.45	0-14700

nae to be dominant over *D pteronyssinus* in only 41% of the houses in Atlanta. They found *D pteronyssinus* dominant in 47% of the houses and the two species to be about equal in number in 12%.

Some sites within a house have more optimal microenvironmental conditions for mite population growth than others, with padded furnishings usually supporting the highest populations.¹⁹ In this regard, the sofa has been found by some investigators to have large mite populations,^{3,6,19} and our study supports this finding. During use, temperature and humidity rises, and the padding allows these conditions to be maintained for a period after use. Additionally, when there is sufficient use, the sofa will collect skin dander and other dust elements.

Another padded furnishing, the bed, has also been shown to harbor large mite populations.^{6,19,21,22} It provides an excellent microclimate for development of mite populations and can sustain them throughout the year, though density fluctuations do occur.² Skin dander accumulates in beds and extended use elevates the temperature and humidity. Coverings on the bed also assist in retaining the humidity and moderating temperature changes. The bed has been implicated as a breeding site^{22,23} and can be a reservoir from which mites spill over into other areas of the house during seasons of peak densities.⁶ The mattress, mattress cover, pillow and bed covering all contribute to the bed's microclimate; however, the mattress appears to be the major infestation site and contains the highest popula-

tions.^{6,12,19-23} The pillow, on the other hand, is found to have the fewest mites.^{12,22} Our study reinforces these findings—pillows yielded little dust and few mites, whereas mattresses yielded large numbers of mites.

Not all studies have implicated mattresses as a primary source of mites. Arlian et al^{2,4} report that carpets and sofas are the principal source of mites in Ohio. The accumulation of dust in carpets supports mite populations well when humidity and temperature are suitable. The most important carpet sites for high mite densities occur in the bedroom and the family room.^{3,6} Our study dealt only with dust samples from carpets beside beds and the mite density levels were of intermediate number in comparison to the other sites investigated.

Any environmental condition affecting humidity, temperature or dust accumulation within the mites' microhabitat should have an impact on mite density. Therefore variables such as type of construction, heating, cooling, ventilation, personal cleaning habits, etc., should be important.^{19,23,24} However, since each home has its own unique combination of physical factors which affect its microhabitats and their microclimates, it is extremely difficult to collect data which statistically demonstrate that such variables have an influence on mite density. Arlian et al^{2,4} found no correlation between mite density and mattress age, mattress use, sofa age, sofa use, bedroom and family room carpet age, sheet change frequency, mattress pad cleaning, house age or

samples, but due to their small numbers, not selected in the subsamples taken to determine pyroglyphid composition. However, in other studies concerning house dust mites in Virginia, *D farinae* and *D pteronyssinus* were also the only pyroglyphids identified.^{14,15}

Although *D pteronyssinus* was found to be dominant in coastal California,¹¹ Atlanta, Georgia,¹³ and Vancouver, British Columbia,¹⁶ in most areas of North America *D farinae* has been found to be the most abundant house dust mite and *D pteronyssinus* the second most abundant.^{4,10,12,17,18} This pattern of abundance was also found in our study, with *D farinae* constituting 70% of all mites sampled and *D pteronyssinus* 28% (2.5:1 ratio). In studies conducted around Charlottesville, Virginia, 120 miles west of Williamsburg, Platts-Mills et al¹⁴ also found members of the genus *Dermatophagoides* to be the most common mite species (95.6%); however, they found *D pteronyssinus* and *D farinae* to occur in approximately equal numbers. In the Hampton Roads area, 50 miles east of Williamsburg, King et al¹⁵ found *D farinae* to be dominant on home ported US Navy vessels (5.8:1 ratio) and *D pteronyssinus* to be dominant in houses (2.9:1 ratio). The reasons for such locality differences remain unknown, although the climate in each of the areas is different.

There is a high incidence of co-existence of *D farinae* and *D pteronyssinus* in the Williamsburg area (21 of 22 houses sampled). A high rate was also recorded for Atlanta, Georgia (17 of 20 houses sampled),¹³ but such is not always the case. In two reports concerning houses from both Dayton and Cincinnati, Ohio, Arlian et al^{2,4} described only seven of 19 (33%) and eight of 18 (44%) houses, respectively, containing both species. In houses inhabited by both species, dominance also varies with locality. We found *D farinae* to be dominant in 16 houses (76%), a percentage very similar to those reported from Ohio (86% and 75%).^{2,4} Smith et al,¹³ however, found *D fari-*

years in residence. Nor did they find mite abundance affected by the frequency of cleaning or the presence or absence of humidifiers. The only significant difference they found related to type of floor covering, with long pile carpets such as shag having larger mite populations than short pile carpets or floors without carpets. The present study also found little effect of physical factors on mite populations; however, contrary to the above study, we did find that sofa use influenced mite populations, but found no effect due to carpet type. It is apparent that a study utilizing larger sample sizes and exercising much greater control over the variables will be necessary to determine which physical factors are important in regulating mite densities.

If is of interest to compare mite densities found in homes in Williamsburg with those found at other localities. Table 3 provides the results of a literature survey which gleaned the highest mean values for mite densities in houses on a worldwide basis. Densities in Williamsburg are quite high compared with other areas of the USA as well as most other areas of the world. The importance of relative humidity on the growth of mite populations has been well established.^{1,2} High humidity throughout the year combined with mild winters are undoubtedly contributing factors to the high mite populations found in Williamsburg houses. Mite counts greater than 300 per gram of dust have been associated with symptoms of asthma.¹⁴ All Williamsburg homes surveyed had populations in excess of this number, and this explains in part the reason for the high incidence of allergy.

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Table 3. Comparison of House Dust Mite Population Densities (mean number of mites per gram of dust) from the U.S.A. and Other Parts of the World.

Location	Mean Number Mites/Gram	Time of Year	Site
Paupa New Guinea ²⁵	1378*	—	Beds
Japan & Taiwan ²⁶	1054	—	Mixed
Iran ²⁴	3293*	Sept	Mixed
Algeria ²⁷	>7000	April	Mattresses
India, Lucknow ²⁸	1986*	—	Unencased Mattresses
Israel, Bat Yam ²⁹	2473	Dec-Aug	Sofas
Switzerland, Basel ²¹	6000-8000	Over year	Mattresses
The Netherlands ³⁰	575**	July	Floors
Belgium, Haverlee ³¹	4597	Feb	Mattresses
France (lowlands) ³²	2120	Sept	Mattresses
Finland ³³	1506*	—	Mattresses
Denmark ³⁴	4080***	Aug	Beds
United Kingdom			
Scotland, Glasgow ²²	1539	—	Beds
Scotland, Orkney (farms) ³⁵	1215	—	Mattresses
England, Birmingham ²⁰	4241*	Aug-Sept	Mattresses
England, London ³⁶	2568	Over year	Mattresses
Barbados ³⁷	4440*	—	Mattresses
United States			
Colorado, Denver ³⁸	240*	Sept	Imported Mattresses
Ohio, Columbus ¹²	382*	Summer	Mattresses
Ohio, Dayton/Cincinnati ²	1110**	June	Mixed
Tennessee, Knoxville ³⁹	343*	—	Mixed
Georgia, Atlanta ¹³	100	Over year	Beds
Virginia, Charlottesville ⁴⁰	897*	—	Mixed
Virginia, Charlottesville ⁷	2000**	Aug	Beds
Virginia, Williamsburg	4580	Aug	Sofas
	4409	Aug	Mattresses
	1483	Aug	Carpets

* estimated from table; ** estimated from graph; *** median

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F. I. Ende, MD, and Mark Ende, MD,
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HUMAN ehrlichiosis caused by an *Ehrlichia canis*-like organism was first described in this country in 1987.¹ Since that time scattered case reports have appeared in the literature, from different states.^{2,3,4} This report deals with two cases in Virginia; one of these two patients may have had Lyme's disease at the same time.

Case Reports

The first patient was a 66-year-old male farmer who was admitted to the Southside Regional Medical Center, Petersburg, on 18 May 1989 with a history of being in excellent health until about one week prior to hospitalization. He complained of being weak, cold, hot, sweating and tremulous. There was no nausea, vomiting or diarrhea. He had noted tremor of his hands about four days prior to hospitalization. He had removed ticks on several occasions during the previous two weeks. On examination he was a well-nourished male who appeared toxic and had a resting tremor. A fine macular rash was noted throughout the trunk, extending even to the scalp. The patient was thought to have Rocky Mountain spotted fever or Lyme's disease and was started on tetracycline and ceftriaxone (Rocephin®). Lumbar puncture was normal, EEG negative, BUN 41, WBC 3,900 with 51% lymphs, platelet count 79,000. Liver function tests on admission were abnormal, AP 123, GGT 72, SGPT 83. Temperature was 102° and became normal in 24 hours. Rash and tremor improved. Agglutination for Rocky Mountain spotted fever was positive 1:256, IGM negative. The test for Lyme's disease was negative. He was discharged on tetracycline. It was felt he should be tested for *Ehrlichia canis* and the results were markedly positive, being 1:4096, IGM was positive >1:20, the latter indicative of a recent infection. Since leaving the hospital the patient has been followed in the office, and two months after the hospitalization

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complains of feeling weak and aching across the chest and shoulders. Followup test for *Ehrlichia canis* was IGG 1:1024, IGM <1:20.

Table 1. Test Results in Patient One

Date	Test	Results
5/18/89	Lyme Disease ABS Quan EIA	Negative
5/20/89	<i>Ehrlichia canis</i> IGG IGM	1:4096 >1:20*
9/21/89	IGG IGM	1:1024 <1:20

*IGM greater than 1:20 means recent infection

The second patient was a 69-year-old male transferred to Southside Regional Medical Center on 30 June 1989 from another hospital. Approximately one week prior to hospitalization the patient became ill, with chills, malaise, fever, nausea and nervousness. His wife stated she had taken ticks off of his back. His past history was significant for a bleeding ulcer and hypertension, and he had been found to have abnormal liver studies prior to his transfer. On physical examination patient's temperature was 102°. General physical exam was negative. Neurological survey done in its entirety was negative. The initial impression was the patient had Rocky Mountain spotted fever, Lyme's disease or ehrlichiosis. Patient was started on Rocephin, one gram daily, and tetracycline. His temperature came down to normal in 48 hours. Studies revealed the blood cultures to be negative, hemoglobin 14.6, WBC 5,200, LDH 568, GGT 56, GOT 86, glucose 232, BUN 38, creatinine 2.6. He had heavy proteinuria. In addition, this patient was found to have a positive anti-HBS and positive HBC, indicating previous hepatitis B infection, but it was felt that this played no part in the present illness. The 24-hour urine collection showed creatinine clearance of 39 cc and 6,300 mg of protein. BUN rose to 54 and creatinine to 3.3, indicating a nephrotic syndrome. Patient showed prompt improvement on the above medications and became greatly desirous of leaving the hospital before results of the studies for various tick diseases were received.

Table 2 shows a rise in titer for ehrlichiosis, confirming the diagnosis of this disease. Also, the titer for Lyme's disease rose from zero to 2.77 index suggesting this patient may also have had this disease.

The patient was readmitted on 15 August 1989 because of severe abdominal pain and discomfort in his legs and arms. Physical examination at this time revealed his abdomen to be tender but there was no rebound. During the course of his hospital stay the pain in the patient's arms and legs became extremely intense. He felt numbness in his feet. His knee jerks were absent. CT scan of the lumbar spine showed spinal stenosis due to a combination of short pedicles

Table 2. Test Results in Patient Two

Date	Test	Results
	Lyme Disease	
6/30/89	ABS Quan EIA	Negative
	ABS IGM Quan EIA	<1.0
7/6/89	ABS Quan EIA	2.77*
	ABS IGM Quan EIA	<1.0
8/15/89	ABS Quan EIA	2.41
	ABS IGM Quan EIA	<1.0
8/24/89	ABS Quan EIA	2.23
	ABS IGM Quan EIA	<1.0
9/15/89	Lyme total antibody	1.59**
	<i>Ehrlichia canis</i>	
6/30/89	IGG	>or = 1:64
	IGM	>or = 1:20
7/6/89	IGG	>or = 1:64
	IGM	>or = 1:20
8/15/89	IGG	>or = 25
	IGM	weakly positive 1:
8/24/89	IGG	1:512
	IGM	<1:20
9/5/89	IGG	1:512
	IGM	<1:20

Four-fold rise in titer diagnostic of disease

*Index = ratio of patient's titer to general population. Index of 2.77 regarded as mid-positive.

**Positive considered to be 1.00

and hypertrophic changes associated with degenerative arthritis in the facets. Myelogram showed a possible herniation of disc at L₄. Serum amylase was 68. BUN was now 8 mg, sed rate 18, stool for blood was negative, amylase clearance was 4.5%, glucose 162, K 3.3, spinal fluid protein was 155 with no cells. EMG revealed findings suggestive of peripheral neuropathy, bone scan was negative. The patient's abdominal pain appeared to subside on its own, leg and arm pain disappeared.

After this hospitalization the patient was followed in the physician's office. He had exacerbation of severe leg and arm pain and was restarted on tetracycline, which appeared to result in a distinct improvement in limb pain. It was felt that the severe pain was due to peripheral neuropathy as reported in previous cases of ehrlichiosis. His proteinuria improved to 1288 on 18 September 1989. Liver studies returned to normal.

Discussion

Ehrlichiosis is a rickettsial leukocytic infection which was first described in dogs in 1935. This disease is often fatal in animals. The vector of this disease is the brown dog tick, *Rhysicephalus sanguineus*. The organisms are obligate intracellular bacteria which infect mononuclear or polymorphonuclear leukocytes. The leukocytic *Rickettsiae* of the genus *Ehrlichia* include the species *E canis*, *E risticii*, *E equi*, *E phagocytophila*, and *E sennetsu*.¹ Cross-agglutination within the genus exist.

The diagnosis of ehrlichiosis is made by a fourfold rise in titer. Identification of the organism can be made

from the infected cells. Electron microscopy shows inclusion bodies in the white cells containing organisms. In both of these cases the patients had left the hospital prior to receiving the report of the elevated serology; therefore, searches for organisms were not done. It is difficult to determine from the literature if cross-reaction titer between *Ehrlichiosis canis* and *Borrelia burgdorferi*, the causative agent in Lyme's disease, occurs, although several cases of cross-reactive serology have been reported, as has possible infection with both organisms.⁷

In humans, ehrlichiosis is manifested by fever, myalgia, nausea, chills, headache. Lab changes are leukopenia, thrombocytopenia, abnormal liver studies. Rash occurs in about 20% of the cases. There has been a report of a possible human fatality.² The drug of choice in treating rickettsial infection is tetracycline. Next drug would be chloromycetin. The drug ceftriaxone, although quite effective in Lyme's disease, is not effective against rickettsial diseases.⁶ Both of the above patients were started on tetracycline along with ceftriaxone. The second patient had moderate increase titer for Lyme's disease. Early treatment can prevent rise in titer in this condition.⁵ The increase in spinal fluid protein is more indicative of Lyme's disease than ehrlichiosis.

Summary

Ehrlichiosis is a disease that should be considered in any case of chills, fever and malaise, with or without a history of tick bites.

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ABSTRACTS

These abstracts are to be presented at the annual meeting of the Virginia Surgical Society on May 4-6 in Hot Springs. Dr. Irving L. Kron is program chairman.

Arterial Bypass Procedures to the Vessels of the Ankle and Foot. Jesse T. Davidson III, MD, Roanoke.

Twenty-nine arterial reconstructive procedures involving vessels of the ankle or foot have been performed in 27 patients since 1985. Sixteen (59%) of patients were men and 11 (41%) were women. Risk factors included diabetes (78%), cardiac disease (37%), hypertension (33%), tobacco use (30%), and cerebrovascular disease (26%). Indications for operation were gangrene (62%), ischemic ulceration (27%), and rest pain (11%). Autogenous tissue was used for all procedures which included *in situ* vein (62%), reverse saphenous vein (14%), combination of *in situ* reversed or translocated vein (16%), lesser saphenous vein (4%), and endarterectomy with vein patch (4%). Distal bypasses were done to the dorsalis pedis artery (62%), posterior tibial artery at the ankle (20%), and distal anterior tibial artery (14%). Proximal anastomoses originated on the common femoral artery (24%), superficial femoral artery (20%), and popliteal artery (48%). There were two postoperative graft failures. One of the failures was revised to femoral-popliteal bypass graft with subsequent limb salvage. Two patients (7%) required below-knee amputations in the postoperative period, one with a patent bypass. Operative mortality was 4%. Cumulative patency at 12 and 24 months was 90%. Autogenous reconstructive vascular procedures at the foot and ankle are effective for limb preservation.

Total Esophagectomy (Blunt) Without Thoracotomy. Juan M. Montero, MD, Chesapeake.

This operation which was first described by W. Denk in 1913 was reintroduced by Dr. George Grey Turner in the 1930s. However, it was not until Dr. Hiroshi Akiyama took up the operation in the 1960s and Dr. Mark Orringer in the 1970s that interest in the rest of the surgical world was rekindled. The technique features an esophagectomy done without a thoracotomy, using blunt dissection through a combined upper midline abdominal and cervical incisions. Following complete thoracic esophageal resection the stomach is pulled up through the posterior mediastinum and cervical esophagogastric anastomosis is carried out. There is a continuing debate as to the rightful historical place for this relatively "new" operation. Sometimes we are slow to adopt useful new operations, which for this particular one is done for benign and malignant lesions.

For this presentation, I will discuss my experience of five patients with cancer of the esophagus operated on between May 1986 and September 1989.

Colonoscopy in Preoperative Evaluation of Patients with Colorectal Cancer. Luiz E.G. Mineiro, MD, Clifton Forge.

One hundred sixty-seven cases of carcinoma of the rectum and colon were retrieved from a total of 2,412 consecutive colonoscopies performed by the author from 1974 to 1988. These data were checked for synchronous and metachronous lesions. All examinations were done at Alleghany Regional Hospital, a 160-bed rural hospital located in western Virginia. Twenty-five (14.9%) synchronous polyps, 15 (8.9%) metachronous polyps, 5 (2.9%) synchronous carcinomas, and 14 (8.3%) metachronous carcinomas were found. Of the 5 synchronous carcinomas, 4 were not suspected. Three of the operations involving these cases were modified accordingly. Five of the metachronous carcinomas were recurrent tumors. Five of the patients with metachronous tumors did not have a colonoscopy at the time they were first treated because either the original operation was performed elsewhere or preceded the collection of these data.

Based on these findings, it appears that preoperative colonoscopy, in conjunction with the abbreviated bowel preparation discussed in this paper, was helpful in the proper planning of the surgical treatment of our patients and might have prevented development of metachronous cancer.

Percutaneous Cholecystostomy. George C. Hoffman, MD, Norfolk.

Cholecystostomy can be an initial lifesaving procedure for patients with acute cholecystitis who are critically ill and at a very high operative risk. We have performed percutaneous cholecystostomy under local anesthesia in four patients with acute cholecystitis who were considered at excessive operative risk because of multisystem failure. One patient with emphysematous cholecystitis and cholangitis also had obstruction of the distal common duct secondary to a pancreatic neoplasm. All four patients' clinical picture improved following drainage of the gallbladder empyema. One patient died of causes unrelated to the biliary tract. One patient has had a subsequent cholecystectomy and two patients are currently being managed as outpatients with cholecystostomy tubes in

place. A review of the literature addresses indications, the diagnostic value, technical aspects, and subsequent outcomes of patients who have undergone this relatively new and innovative method of gallbladder decompression.

Complications of Dialysis Access—A Six-Year Study. Stephen L. Hill, MD, and Antonio T. Donato, MD, Roanoke.

Chronic renal failure in most patients is permanent, with no hope of transplant, and thus dialysis access problems are chronic and potentially life-threatening complications. We looked at our complications with dialysis access procedures over a six-year period. In this period we had 76 patients and performed 158 procedures. Our first option was always a Cimino fistula. Only 10 patients (13%) were candidates for a Cimino fistula. There were two failures in this group with two other patients requiring revision of an anastomosis two years later. There were no other complications with these fistulae and they continue to function.

The vast majority of the dialysis access procedures were upper arm grafts with reversed tapered PTFE. There were 73 patients in this group and required a total of 147 procedures to keep the grafts patent for the study period or until death. As to be expected, the primary complications were thrombosis and infection. There were no patients with access induced congestive heart failure or symptomatic steal syndrome.

The major complication was thrombosis. There were 24 patients (31%) who required revision, thrombectomy or placement of a new graft over the six-year period. This entailed a total of 57 procedures. In only one patient was a simple thrombectomy done. All other patients required either a major revision with an anastomosis to a new vein or placement of an entirely new graft. The thromboses were almost exclusively caused by obliteration of the venous lumen with pseudointimal hyperplasia. The other major complication was infection. There were 8 patients (10.5%) with an infected graft. This necessitated two procedures: one to remove the graft and repair the artery with an autogenous vein patch, the other to place a new graft in the other extremity. There was a small subset of 5 patients responsible for many of the complications of infection and thrombosis and for over 45% (37) of reoperative procedures in the entire population.

The complications of dialysis are tedious, recurrent, and complex but there are several lessons we have learned. 1) Infected grafts will always have to be removed and require two operative procedures. 2) Thrombosed grafts can only rarely be thrombectomized successfully; more often they will require a major revision of the venous anastomosis or an entirely new graft. 3) A small subset of the dialysis population is not amenable to vascular access due to

recurrent thrombosis and/or infection. 4) An autogenous fistula should be placed whenever possible even, if necessary, after a prosthetic graft has already been placed for immediate dialysis.

Use of the Supraclavicular Approach for Surgical Treatment of Thoracic Outlet Syndrome. Stephen W. Ely, MD, Don E. Detmer, MD, Irving L. Kron, MD, and Thomas M. Daniel, MD, Charlottesville.

The present study evaluated the effectiveness of the supraclavicular approach for the surgical treatment of thoracic outlet syndrome (TOS). 21 sequential cases treated at the University of Virginia from July 1986 to December 1989 were retrospectively reviewed. This group included 18 females and 3 males, ages 22 to 44. Through a supraclavicular incision, the following procedures were performed: cervical rib resection (33%), 1st rib resection (68%), anterior scalenectomy (95%), middle scalenectomy (48%), subclavian artery aneurysm repair (5%), and cervical sympathectomy (5%). The mortality rate was 0% and the complication rate was 24%. This included 2 patients with pleural effusions, 1 with pneumothorax, 1 with causalgia, and 1 with a stretch injury of the brachial plexus. There were no vascular complications. All complications were temporary except for the brachial plexus injury which had a 90% recovery at 2 years. Followup was available for 100% at a mean interval of 19.1 months. Excellent relief of symptoms was achieved in 75% initially, but 5 of these 16 patients developed mild to moderate recurrent symptoms in the followup period. 10% showed improvement in their symptoms following surgery and 10% showed no change from their preoperative state. 1 patient (5%) had a worsening of symptoms following surgery and that was the patient with the brachial stretch injury.

This study demonstrates that the supraclavicular approach for TOS allows for the versatility necessary in treating the many possible causations of this syndrome. 85% of the patients were improved by the surgery. The absence of any objective test to diagnose TOS continues to make treatment of this problem a diagnostic and therapeutic challenge.

Clinicopathologic Review of Distal Common Bile Duct Cancers. George A. Parker, MD, Michael Bousamra, MD, and A. Scott Mills, MD, Richmond.

To define the natural history of cancers arising in the distal common bile duct (CBD) a clinical and pathologic review of 22 patients undergoing potentially curative resections was done. Operative mortality was 22%, however there were no operative deaths in the 13 patients treated after 1970. Complications included intraperitoneal abscess in 7 patients, pancreateojunostomy leak in 3 patients, and enterocutaneous fistulae in 2 patients. Nineteen tumors were either well differentiated (WD) or moderately well differentiated (MWD), 2 were WD or MWD, while 1 was poorly

differentiated. Eight patients had nodal metastases, while 14 did not. The degree of differentiation did not correlate with the presence of nodal metastases. Perineural invasion as well as pancreatic or duodenal invasion occurred in 19 patients. In only 2 instances was the tumor confined to the CBD. Excluding operative deaths the median survival was 22 months. The patients with nodal metastases had a mean survival of 20 months, while those without nodal spread had a mean survival of 33 months. Three years post operation only 3 patients were free of disease: 2 had no invasion of the pancreas or duodenum, and 1 had minimal invasion of the pancreas. We concluded that favorable prognostic factors for resected distal CBD tumors include lack of invasion of surrounding structures and absence of nodal metastases. Degree of differentiation did not correlate with survival or presence of nodal metastasis. Finally, long-term survival was limited to patients with no or minimal involvement of adjacent structures.

Management of Locally Advanced Breast Cancer with Multimodality Therapy. James L. Frank, MD, and Barry D. Bear, MD, Richmond.

Optimal management of locally advanced breast cancer is controversial. Claims of superiority for neoadjuvant systemic therapy are based on comparisons with outdated historical control groups who received no chemotherapy. Between 1978 and 1987, 119 patients with locally advanced breast cancer underwent primary treatment and follow up at the Medical College of Virginia. Clinical substaging included 63 (52%) patients with stage IIIA, 43 (36%) patients with stage IIIB non-inflammatory, and 13 (11%) patients with stage IIIB inflammatory disease. Initial treatment consisted of local therapy in 108 (91%) patients; 11 patients initially received systemic therapy. Fifty-three (49%) patients received conventional systemic adjuvant therapy after local treatment. Median follow up is 44 months (range 3-119 months). Actuarial five year survival for the entire group is 53%. This compares favorably with recent series using neoadjuvant chemotherapy in which 5-year survival rates of 40 to 65% have been reported. Significant prognostic indicators for survival include the percentage of pathologically involved lymph nodes and the presence of an inflammatory component. Steroid receptor status, age, local grave signs, tumor size, and systemic adjuvant therapy did not significantly alter survival. This series represents a contemporary control group of patients with locally advanced breast cancer in whom conventionally accepted guidelines for local and systemic adjuvant therapy were used. Until the optimal sequence of therapy is determined by prospective randomized trials, series such as this may serve as more appropriate controls to which the results of new therapies should be compared.

Gallstone Lithotripsy: Improved Results with Adjuvant Bile Salt Therapy. Bruce D. Schirmer, MD, Daniel J. Pambianco, MD, Janet Dix, PA-C, Sarah Fabian, RN, Patricia Abbott, MD, Jay Gillenwater, MD, and R. Scott Jones, MD, Charlottesville.

Symptomatic patients with ≤ 3 non-calcified gallstones and a functioning gallbladder were randomized as part of a prospective double-blind national study to determine the role of adjuvant bile salt therapy in conjunction with extracorporeal shock wave lithotripsy (ESWL) for gallstones. The 31 patients, 22 women and 9 men, average age 50, underwent ESWL treatment using the Dornier MPL 9000 lithotripter. The mean stone volume present prior to treatment was similar for patients receiving ursodeoxycholic acid (URSO, n = 11) and placebo (PLAC, n = 20), at $2.68 \pm 2.19 \text{ cm}^3$ and $3.58 \pm 3.63 \text{ cm}^3$ respectively. Fourteen patients in the PLAC group and 6 in the URSO group had solitary gallstones. All treatments required only intravenous sedation as anesthesia. Patients received 1500 shock waves at an average of 18 kV. Six weeks post treatment patients with ultrasound-documented fragments $\geq 5 \text{ mm}$ underwent a second similar ESWL treatment. Nine of 20 PLAC patients and 7 of 11 URSO patients required second treatments. At six months follow-up for all patients 50% (3/6) of URSO patients and 7.1% (1/14) of PLAC patients with initial solitary gallstones were stone and fragment-free ($p < .05$). No patient with multiple stones in either group was stone-free. Patients tolerated the treatments well, but biliary colic, usually mild, was reported following 78.7% of treatments. Only two adverse complications occurred: one patient developed pancreatitis that resolved with conservative care and one patient developed transient jaundice; both patients later had elective cholecystectomy. We conclude that ESWL treatment for gallstones is safe but the efficacy may be less than that reported elsewhere. Adjuvant bile salt therapy appears to improve results in patients with solitary gallstones.

A Single-Center Experience with Pancreas Transplantation: Results and Expectations. Timothy L. Pruitt, MD, and John B. Hanks, MD, Charlottesville.

Clinical pancreas transplantation is a therapy which is evolved to relieve Type I diabetics from the need for exogenous insulin, maintain euglycemia and hopefully show the secondary complications of diabetes. Although the early results of clinical pancreas transplants were poor, recent changes in technique and immunosuppression has led to better long-term results. At the University of Virginia, six pancreas allografts have been performed since April 1988, five in combination with kidney transplant and one pancreas allograft alone. All grafts are working, with maintenance of fasting euglycemia and normal hemoglobin A1C levels. Two surgical complications have

occurred, one late rupture of the bladder (six months post transplant) and bleeding from the donor duodenum which has required cystoscopy.

Preliminary evaluation of carbohydrate metabolism in these patients has revealed fasting hyperinsulinemia (in excess of 15 μ units/ml with a concomitant blood sugar of 70 to 80 mg/dl) and marked hyperinsulinemia in response to a glucose challenge. There is substantial evidence that carbohydrate metabolism is not normal in spite of the superficial appearance of being "cured"

of their diabetes. Metabolic complications have included acidosis and intermittent hyperkalemia presumed secondary to cyclosporine A as well as complicated by the acidosis. Pancreas transplantation is a feasible and reproducible operation to ameliorate the need for exogenous insulin for the Type I diabetic. Its long-term efficacy and benefit for these individuals remains to be discerned. The advent of combined pancreatic and renal transplantation has yielded reproducible results.

Point of View: The Case for Economy Class

AS THE NATION struggles to contain soaring health care expenditures, the decade of the nineties belongs to the nursing profession. Nurses are in short supply, as physicians were during the sixties and seventies. Hospitals compete with higher salaries—for the betterment of health care, in my opinion. Attrition in the nursing profession should decline, as well as the taking of secondary jobs to supplement income.

In the present climate, hospitals cannot function with fewer nurses (though they can get along with fewer physicians). This is evident in some of the hospitals in Hampton Roads, which have closed patient rooms for lack of nurses.

What I can't understand is why hospitals continue to expand the number of private-room beds, knowing the critical nursing situation. What is worse is the total demise of hospital wards. Why is this so, when business sense would seem to dictate that ward beds, which need fewer nurses, are most

cost effective? The answer: Wards are looked down on in these times of affluence. "I sure don't want to be known as the administrator of the hospital with wards," quipped one administrator recently.

So there we are. We can no longer afford health care because we have become spoiled by the hospitals' affluence. Everything is first-class—private room with color TV and a telephone. It is time to educate the American public not to equate luxury with quality of care. No health care professional is going to withhold or modify treatment just because the patient is on a ward. And there should be no stigma attached to being on a ward. I always buy an economy-class airplane ticket, and I still arrive at my destination the same time as the guy in the first-class section who was served cocktails as he sat in a wider, more comfortable seat.

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VIRGINIA MEDICAL

The Price of Regulating

THE conflict between the rights of society and the rights and freedom of action of the individual is with us always and is well illustrated in the new requirements discussed in the article on waste disposal in this issue.

Our office has its own waste dumpster, and for years everything was lumped together and dumped therein. We started getting letters from the waste removal company indicating that we should not put sharp objects, specifically plastic syringes with needles, in the dumpster. Finally a letter informed us that it was a federal offense to dispose of needles with the other trash and that we would be fined if we continued to do so. We then bought a supply of the expensive red

puncture-resistant containers and arranged to have these incinerated—at an additional price in time and money.

The local hospital spends six to ten times as much to dispose of sharps and waste termed “infectious” as it does for regular waste disposal.

Even though, as indicated in the article, there have been no public health problems associated with the previous methods of disposal of these wastes, we are now shouldered with these rules. We can and will obey the law, but the increased cost will necessarily, as always, be passed on to the public.

RAYMOND S. BROWN, MD

The State Board of Medicine

As the saying goes, “It ain’t what it used to be.” In this case, reference is made to the State Board of Medicine. When this writer joined the Board in 1976, approximately one-half day of the three-day session was devoted to the impaired physician. Ten years later three-quarters of each entire session dealt with that problem.

During the latter part of that particular ten-year period and culminating in 1985 the State Board of Medicine was the object of multiple criticisms; as stated at that time, the criticisms were:

1) The impaired physician is not recognized; when recognized, he is not reported; when reported, he is not disciplined; and when discipline is accomplished,

the process is painfully slow and the physician is often allowed to continue practice for a variably lengthy period of time.

2) The State Board of Medicine is composed of physicians; they are loathe to regulate and discipline their colleagues. There should be public representation on the board.

3) There is no effort to elicit complaints; when complaints are filed, screening of these complaints is accomplished by a single member of the board, and all decisions as to the need for further consideration and/or investigation are made by this member.

Beginning in 1986, all of these criticisms have been answered, some more effectively than others.

Whenever a physician is disciplined by a hospital, this disciplinary measure must be reported to the State Board of Medicine. While the celerity of the process still is the object of some criticism, that, too, has been improved as much as possible. Little can be done about the delay caused by due process of law.

The State Board of Medicine proposed the appointment of one public member. Two were appointed. While there is no real objection to the additional public member, it was hardly the idea of the Board that both appointees would be lawyers. However, these appointments have largely quieted this criticism.

Beginning in 1986, complaints have been solicited, encouraged and facilitated by the provision of a toll-free telephone number. In 1985 there were 258 complaints; by July 1988-89 the number had risen to 371. However, there does appear to be a levelling-off of the curve.

During the period 1967 to 1986 Dr. George Carroll served as secretary of the Board and performed the original screening; in the opinion of most, he did it well. In 1987 the position of medical director of the board was established and Dr. Hilary Connor was appointed to the post. There is no question that a competent full-time director can be more effective than one who serves on a part-time basis and Dr. Connor has acquitted himself well. He, along with the president of the Board, now does the screening. Before a case is permanently closed without a hearing, that case is reviewed by the Board. If the Board does not agree, the case is reopened.

The medical profession in Virginia appears to be well regulated. Now, if certain other professions . . .

E.L.K., JR.

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VIRGINIA MEDICAL OBITUARY

• **Chapman Hunter Binford, MD**, Arlington; Medical College of Virginia, 1929; age 90; died February 9, 1990. Dr. Binford was widely respected for his research in pathology, notably at the Armed Forces Institute of Pathology in Washington.

• **Francis J. Clements, MD**, Yorktown; Medical College of Virginia, 1923; age 93; died January 2, 1990. Originally a general practitioner in Fluvanna County, Dr. Clements worked for the State for 29 years as director of local health departments in Dinwiddie, Mecklenburg, Prince Edward, and Sussex Counties.

• **Robert G. Kindred, MD**, Rockville, Maryland, orthopedic surgeon; University of Virginia School of Medicine, 1948; age 64; died of leukemia in Baltimore on April 27, 1989.

• **Carroll H. Lippard, MD**, Lynchburg obstetrician and gynecologist; University of Pennsylvania School of Medicine, 1946; age 66; died February 13, 1990. He had been president of the Lynchburg Academy of Medicine and the State Board of Medicine.

Loomis Anti-tuberculosis Sanatorium in Liberty, New York, and after arriving in Norfolk became chairman of the Anti-tuberculosis League, participating actively in related clinical research and educational activities. He was a charter member and a fellow of the American College of Chest Physicians and, along with Dr. C. Lydon Harrell, became recognized as a specialist in chest disease.

He witnessed the birth of the therapeutic era of medicine and greeted each new therapeutic modality with enthusiasm, frequently participating in clinical trials. He published papers on his clinical experience with sulfapyridine and sulfathiazole in the treatment of staphylococcal infection and made observations of the potential for these drugs in the prevention of rheumatic heart disease.

The field of cardiac medicine was in its infancy during Bryan's early years. The EKG was invented in 1903 and was not in common use. Myocardial infarction was more frequently recognized after Herrick's description in 1912, and Bryan's interest prompted him to read one of the early papers on coronary occlusion before the Seaboard Medical Association in 1951. He later presented a layman's version of the same paper to the Kiwanis Club in 1957. He became the founding president of the Tidewater Heart Association and played an active role as well at the state and national levels.

Paralytic poliomyelitis was epidemic in the Forties and early Fifties and Bryan, of course, was active in its treatment. In an effort to enlist community support, he became actively involved in the Norfolk City and County Chapters of the National Foundation for Infantile Paralysis.

Few diseases escaped his interest. The management and treatment of diabetes was new, and Bryan played an active role in educating the profession about diabetes and became a member of the American Diabetic Association. In 1950, he presented a paper on "The Pregnant Diabetic" before the Seaboard Medical Society.

Bryan was a life-long member of the AMA and The Medical Society of Virginia. He also strongly supported the Norfolk County Medical Society and served one year as its president. Early in his career he became a diplomate of the American Board of Internal Medicine and, shortly thereafter, attained the status of fellow of the American College of Physicians. He helped organize and became president of the fledgling Virginia Society of Internal Medicine, an organization dedicated to influencing health and social policy. Bryan always had strong views about access to health

Memoir of R. B. Grinnan, Jr. 1908-1989

By John Franklin, MD, Samuel McDaniel, MD,
Frederick Woodson, MD, and Harry Wise, MD

The son of Presbyterian missionaries whose pastorates extended from Japan to North Carolina, Randolph Bryan Grinnan, Jr., MD, was born in Henderson, North Carolina, but spent the major part of his life in Virginia. He graduated from Maury High School and attended the University of Virginia for his undergraduate and medical school education, receiving his MD degree in 1932. He interned at the Boston City Hospital and served his residency at Doctor's Hospital in New York City. He established his practice in Norfolk thereafter and practiced medicine continuously until his retirement in October 1978.

During his 50 years of practice, Bryan witnessed many changes in the field of medicine, the pattern of diseases, and the causes of death. Respiratory diseases, particularly tuberculosis, were the major killers during the early years of his career, and he positioned himself to play an active role in their prevention and control. He served one summer on the staff of the

care and was as concerned about the distribution of health care as about the health of his community.

He served for many years on the Advisory Commission to the Director of Human Resources in Norfolk and was a member of the Norfolk Hospital Association, where he was able to influence indigent health care. Early on, he strongly advocated a free-standing Public Health Department separate from the Department of Human Resources. When this separation was effected, he served as chairman of the Advisory Commission to the new department. In 1988, he received the first Distinguished Service Award for 40 years of service to the department.

Bryan was always interested in the dissemination of medical knowledge to the profession and to allied health personnel. He frequently participated in the training of nurses, published articles in the VIRGINIA MEDICAL MONTHLY and presented papers to local medical societies. He helped organize a Journal Club for internists and served on the editorial board of a local publication called *The Internist*. He was an active staff member of DePaul and Leigh Memorial Hospitals and served as chief of the Norfolk General Hospital Medical Department. He also served as civilian consultant to the Portsmouth Naval Hospital from 1947 to 1960.

Throughout his life, Bryan loved and enjoyed the outdoors, sharing his extensive knowledge of sailing, hunting and fishing with his three sons. On board his catboat, "The Tempest," they took frequent trips to the Eastern Shore of Virginia and to coastal North Carolina. In his later years, sensitive to the diminishing quality of our environment, his interests turned toward conservation, and he contributed significantly to the efforts of several area organizations devoted to environmental concerns.

Bryan and his wife, Adelaide Richardson Grinnan, have left a significant legacy to us all: a tradition of excellence in medical practice; concern for our people, our community, and our environment; as well as three sons, R. Bryan Grinnan, III, an officer with Sovran Bank in Norfolk; George L.B. Grinnan, MD, a cardiac surgeon in Norfolk; and Richardson Grinnan, MD, chief healthcare management officer, Blue Cross/Blue Shield of Virginia, Richmond. The contributions his sons make to our communities are indicative of Bryan's success as a father and honor his memory.

His last years of practice were spent as a charter member of the newly organized Norfolk Diagnostic Clinic, from whence he retired in October 1978. He continued to play an active role in many of his professional associations after his retirement until his health failed in the past two years, and his long and brilliant career ended in his 81st year on February 20, 1989.

Bryan is survived by his wife of 55 years, his three sons, seven grandchildren, and one great grandchild. Those of us who knew him as a friend and a professional colleague salute him with this tribute.

Memoir of John Lynch 1909-1989

By William H. Harris, Jr., MD,
Thomas W. Murrell, Jr., MD, and
E. Randolph Trice, MD

John Phillip Lynch was born June 13, 1909, and died July 15, 1989.

Although a native of New Jersey, he was reared in St. Petersburg, Florida, where he graduated from high school in 1926. He continued his education at Washington and Lee University where he received an AB degree summa cum laude in 1930. He was elected to Phi Beta Kappa and was the recipient of the prestigious Algernon Sydney Sullivan Award. His medical degree was earned at the Medical College of Virginia in 1935, where he became a member of Alpha Omega Alpha Honor Medical Society. Dr. Lynch trained at



JOHN PHILLIP LYNCH, MD
1909-1989

the University of Wisconsin and Medical College of Virginia Hospitals. He joined the staff of the McGuire Clinic-St. Luke's Hospital in 1937. Here he remained in the active practice of internal medicine and geriatrics until his retirement in 1984. He was chief of the department of medicine at St. Luke's Hospital from 1962 to 1974 and vice president of St. Luke's Hospital Corporation in 1965.

Throughout his career he taught students at the Medical College of Virginia, where he was on the clinical faculty and from 1953 to 1974 held the rank of assistant clinical professor of medicine. For more than 40 years Dr. Lynch taught student nurses at St. Luke's Hospital. In 1988 he wrote a history of the Nursing School entitled, "One Hundred Fruitful Years."

Dr. Lynch was a man of strong convictions and

superb judgment who was outspoken in support of the causes he espoused and unafraid to express his opinion on controversial matters relating to medical practice. His activities included involvement in local, state, and national medical organizations. He served on many committees in the Richmond medical community.

Dr. Lynch was elected president of the Richmond Academy in 1962 and served as chairman of the board the following year. He championed the cause of medical education and cautioned against too much emphasis on research. He was deeply concerned about the practice of medicine in this country and the training of students in our medical schools. At an Academy meeting he said, "There are many of us who feel that the emphasis on research is being overdone to the detriment of training young men to look after the sick of our communities."

Dr. Lynch was an expert clinician with a broad knowledge of internal medicine who was greatly concerned about the care of the elderly. He believed that older patients need intelligent understanding by physicians as well as therapeutic expertise. He was chairman of the Committee on Aging and Chronic Illnesses of the Medical Society of Virginia from 1961 to 1964 and a member of this Committee from 1979 to 1984. He chaired the Community Study Task Force on Aging for the City of Richmond in 1974. For many years he was an active member and officer of First Presbyterian Church.

Dr. Lynch contributed many papers to various medical journals, primarily **VIRGINIA MEDICAL**.

His professional memberships included the Richmond Academy of Medicine, Richmond Society of Internal Medicine, Medical Society of Virginia, American Medical Association and the Caduceus Club. He was a diplomate of the American Board of Internal Medicine and a fellow of the American College of Physicians and the American Geriatric Society.

Following his retirement in 1984, he wrote a history of the McGuire Clinic entitled, *The McGuire Clinic: the First Sixty Years (1923-1983)*, which was published in 1987.

This tribute from his McGuire Clinic associates accurately describes the personality and character of John Phillip Lynch:

The McGuire Clinic mourns the passing of one of its leading physicians, shining intellects and history makers. We will miss his sparkling wit, inquisitive mind and his presence. He shared his talents with many committees in the community and on behalf of the medical profession. Dr. Lynch left a legacy we are proud to be part of and anxious to perpetuate.

Dr. Lynch was a devoted husband and father. He is survived by his wife, Helen Davis Lynch, his daughter, Sally Guy Brown, and two grandchildren.

Memoir of J. E. Warren 1915-1989

By William S. Foreman, Jr., MD

Dr. Joseph Edwin Warren, a highly respected obstetrician and gynecologist in Lynchburg, died on Sunday, May 28, 1989.

Born in Hartwell, Georgia, on October 8, 1915, he was the son of the late Mr. and Mrs. Mark T. Warren. Dr. Warren received his premedical education at the University of Georgia in Athens and his medical degree from the University of Georgia School of Medicine in Augusta in 1938. He was a member of the Alpha Omega Alpha honorary medical fraternity. He was licensed to practice medicine in Georgia in 1938.

He served an internship and residency in obstetrics and gynecology at Charity Hospital in New Orleans, Louisiana, from 1938 until 1942. In August 1942 he entered the service of the US Army Air Force and served as a flight surgeon in the Mediterranean theater, attaining the rank of major. For his service he was awarded the Bronze Star. Major Warren was discharged in November 1945.

He was an instructor in obstetrics and gynecology at Louisiana State University from January 1946 through March 1946 and therefore was licensed to practice in Louisiana as well as Georgia and Virginia. He began the private practice of obstetrics and gynecology in Lynchburg in May 1946, where he continued to have an open ear and heart for his many devoted patients until retirement.

Dr. Warren was a member of the American Medical Association, a fellow of the College of Obstetrics and Gynecology, a diplomate of the American Board of Obstetrics and Gynecology and a member of The Medical Society of Virginia, the American Medical Association, the South Atlantic Association of Obstetricians and Gynecologists, and the staffs of Virginia Baptist and Lynchburg General-Marshall Lodge Hospitals. He was a member of the First Presbyterian Church.

He is survived by his wife, Anne Comolli Warren, one son, Joseph Alex Warren of Lynchburg, and two grandsons.

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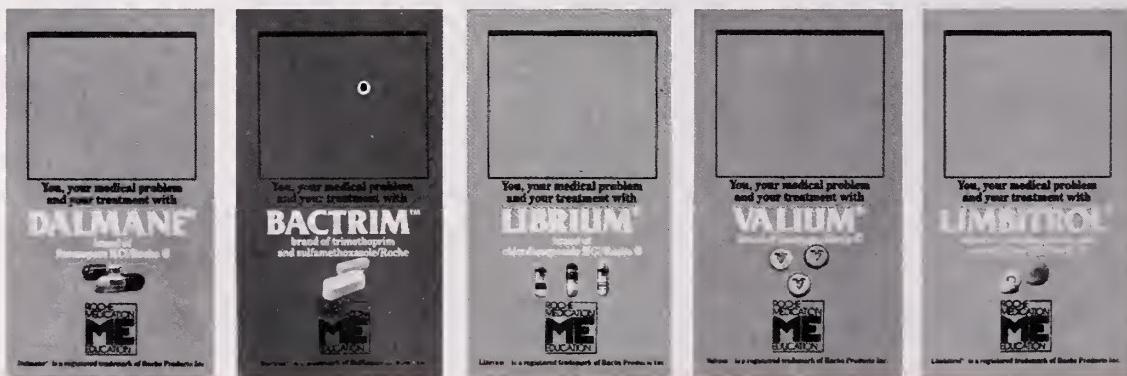


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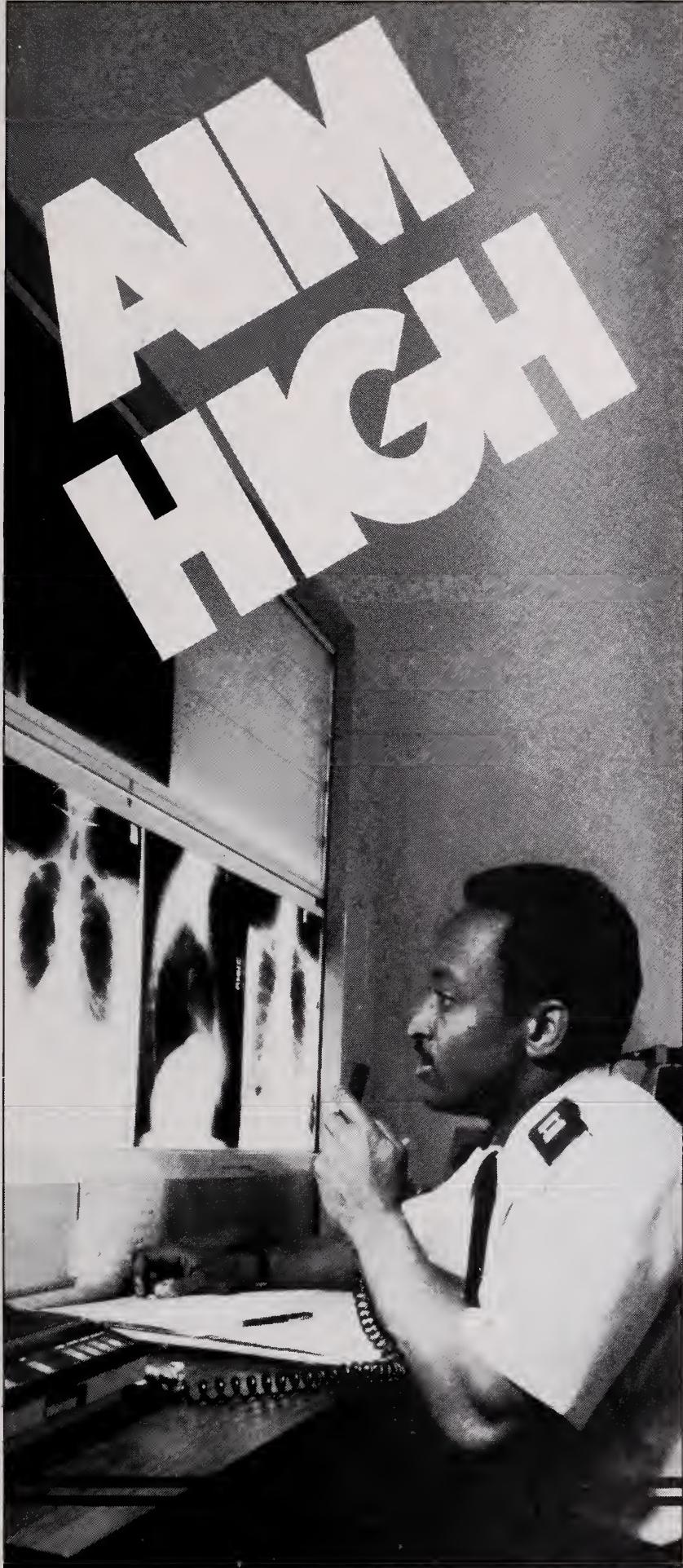
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Annual Meeting of the Virginia Chapter, American College of Radiology, the Homestead, Hot Springs. Patricia R. Berry, 703-669-8312.

May 4

Reimbursement Seminar: Getting Your Fair Share (Virginia Chapter, American College of Emergency Physicians), Fredericksburg. Gwen E. Messler Harry, 804-966-5966.

May 4-6

10th Annual Clinical Concerns in Primary Care (Medical College of Virginia/VCU), Williamsburg. CME Office, 804-786-0494.

May 5

Nutritional Support of the Trauma Patient (Fairfax Hospital), Falls Church. Cathy Sarraf, 703-698-2572.

May 8

8th Annual Ethics Symposium: The Dilemma of Fetal Viability—Medical and Legal Impact on the Delivery of Health Care (Fairfax Hospital), Falls Church. Cathy Sarraf, 703-698-2572.

May 10-13

Annual Meeting, Virginia Orthopaedic Society, Wintergreen. Donna Scott, 804-353-2721.

May 14-18

Topics in Clinical Medicine (Johns Hopkins), Baltimore. 40 credit hrs. Fee, \$650. CME Office, 955-2959.

May 25-27

Advances in Pediatrics (American Academy of Pediatrics), Hilton Head Island, South Carolina. 16 credit hrs. CME Registration, 800-421-0589.

May 26-28

11th Annual Urogynecology and Pelvic Surgery (Medical College of Virginia/VCU), Williamsburg. CME Office, 804-786-0494.

May 29-June 1

19th Annual Educational/Scientific Symposium (Society of Critical Care Medicine), San Francisco. 34 credit hrs. Lisa Parks, 714-870-5243.

June 3-10

Reproductive Endocrinology and General Gynecology (Johns Hopkins), Hilton Head Island, South Carolina. 33 credit hrs. Office of CME, 301-955-2959.

June 4-8

6th Edition, Family Medicine Review Course (Eastern Vir-



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June 4-8

General Surgery Seminar: Update and Review (Lloyd Noland Hospital), *Hilton Head Island, South Carolina*. CME Office, 205-783-5276.

June 6-10

7th Annual Meeting of the Southern Orthopaedic Association, Maui, Hawaii. Kathy McLendon, 205-945-1848.

June 7-9

Update in Obstetrics (American College of Obstetricians/Gynecologists), *Norfolk*. 16 credit hrs. Fee, \$515. ACOG Registrar, 800-673-8444.

June 7-10

14th Annual Postgraduate Course on Rehabilitation of the Brain-Injured Adult and Child (Medical College of Virginia/VCU), *Williamsburg*. CME Office, 804-786-7290.

June 11-15

Adult Infectious Disease Seminar: Current Update (Lloyd Noland Hospital), *Hilton Head Island, South Carolina*. CME Office, 205-783-5276.

June 14-16

35th Annual Great Smokey Mountains Pediatric Seminar (University of Tennessee), *Gatlinburg, Tennessee*. Abby Hall, 615-544-9190.

June 15-16

Arrhythmias: Interpretation, Diagnosis and Management (Medical Education Resources), *Williamsburg*. Deborah Helton, 800-421-3756.

June 20-23

Second Baltimore Perinatal Colloquium (Johns Hopkins/University of Maryland), *Baltimore*. 24 credit hrs. Office of CME, 301-955-2959.

June 21-24

Annual Meeting, Virginia Society of Ophthalmology, Norfolk. Donna Scott, 804-353-2721.

June 22-24

Clinical Pediatrics (American College of Pediatrics), *Washington, DC*. 16 credit hrs. CME Registration, 800-421-0589.

June 24-27

17th Annual Harvard Medical School Course on Interstate Care Medicine: The Art and Science of Critical Care, Boston Massachusetts. Bart Chernow, MD, 617-726-2858.

June 26-30

Family Practice Seminar: Update and Review (Lloyd Noland Hospital), *Hilton Head Island, South Carolina*. CME Office, 205-783-5276.

JUNE 16

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- July 8-11, Addiction Medicine, 14 hrs, \$360
- July 9-10, Child Abuse, 12 hrs, \$150
- July 9-13, Allergy/Asthma/Dermatology, 18 hrs, \$400
- July 9-13, Pediatrics, 16 hrs, \$400
- July 15-17, Gastroenterology, 12 hrs, \$300
- July 17-20, Urology, 15 hrs, \$375
- July 17-20, Surgical Techniques, 15 hrs, \$400
- July 22-26, Ophthalmology, 18 hrs, \$450
- July 23-27, Anesthesiology, 15 hrs, \$375
- July 29-August 3, Obstetrics/Gynecology, 22 hrs, \$500
- July 30-August 2, Diabetes, 15 hrs, \$375
- July 30-August 3, Otolaryngology, 18 hrs, \$450
- August 12-16, Emergency Medicine, 24 hrs, \$400
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Description: Yohimbine is a 3a-15a-20B-17a-hydroxy Yohimbine-16a-carboxylic acid methyl ester. The alkaloid is found in Rubaceae and related trees. Also in Rauvolfia Serpentina (L) Benth. Yohimbine is an indolalkylamine alkaloid with chemical similarity to reserpine. It is a crystalline powder, odorless. Each compressed tablet contains (1/12 gr.) 5.4 mg of Yohimbine Hydrochloride.

Action: Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalamic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

Indications: Yocon® is indicated as a sympatholytic and mydriatic. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral α-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.^{1,2} Also dizziness, headache, skin flushing reported when used orally.^{1,3}

Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.^{1,3,4} 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.³

How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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VASOTEC®

(ENALAPRIL MALEATE | MSD)

VASOTEC is available in 2.5-mg, 5-mg, 10-mg, and 20-mg tablet strengths.

Contraindications: VASOTEC® (Enalapril Maleate, MSD) is contraindicated in patients who are hypersensitive to this product and in patients with a history of angioedema related to previous treatment with an ACE inhibitor.

Warnings: Angioedema. Angioedema of the face, extremities, lips, tongue, glottis, and/or larynx has been reported in patients treated with ACE inhibitors, including VASOTEC. In such cases, VASOTEC should be promptly discontinued and the patient carefully observed until the swelling disappears. In instances where swelling has been confined to the face and lips, the condition has generally resolved without treatment, although antihistamines have been useful in relieving symptoms. Angioedema associated with laryngeal edema may be fatal. **Where there is involvement of the tongue, glottis, or larynx likely to cause airway obstruction, appropriate therapy, e.g., subcutaneous epinephrine solution 1:1000 (0.3 mL to 0.5 mL), should be promptly administered.** (See ADVERSE REACTIONS.)

Hypotension: Excessive hypotension is rare in uncomplicated hypertensive patients treated with VASOTEC alone. Patients with heart failure given VASOTEC commonly have some reduction in blood pressure, especially with the first dose, but discontinuation of therapy or continuing symptomatic hypotension usually is not necessary when dosing instructions are followed; caution should be observed when initiating therapy. (See OOSAGE AND ADMINISTRATION.) Patients at risk for excessive hypotension, sometimes associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death, include those with the following conditions or characteristics: heart failure, hyponatremia, high-dose diuretic therapy, recent intensive diuresis or increase in diuretic dose, renal dialysis, or severe volume and/or salt depletion of any etiology. It may be advisable to eliminate the diuretic (except in patients with heart failure), reduce the diuretic dose, or increase salt intake cautiously before initiating therapy with VASOTEC in patients at risk for excessive hypotension who are able to tolerate such adjustments. (See PRECAUTIONS, Drug Interactions, and ADVERSE REACTIONS.) In patients at risk for excessive hypotension, therapy should be started under very close medical supervision and such patients should be followed closely for the first two weeks of treatment and whenever the dose of enalapril and/or diuretic is increased. Similar considerations may apply to patients with ischemic heart disease or cardiovascular disease in whom an excessive fall in blood pressure could result in a myocardial infarction or cerebrovascular accident. If excessive hypotension occurs, the patient should be placed in the supine position and, if necessary, receive an intravenous infusion of normal saline. A transient hypotensive response is not a contraindication to further doses of VASOTEC, which usually can be given without difficulty once the blood pressure has stabilized. If symptomatic hypotension develops, a dose reduction or discontinuation of VASOTEC or concomitant diuretic may be necessary.

Neutropenia/Agranulocytosis: Another ACE inhibitor, captopril, has been shown to cause agranulocytosis and bone marrow depression, rarely in uncomplicated patients but more frequently in patients with renal impairment, especially if they also have a collagen vascular disease. Available data from clinical trials of enalapril are insufficient to show that enalapril does not cause agranulocytosis at similar rates. Foreign marketing experience has revealed several cases of neutropenia or agranulocytosis in which a causal relationship to enalapril cannot be excluded. Periodic monitoring of white blood cell counts in patients with collagen vascular disease and renal disease should be considered.

Precautions: General. Impaired Renal Function: As a consequence of inhibiting the renin-angiotensin-aldosterone system, changes in renal function may be anticipated in susceptible individuals. In patients with severe heart failure whose renal function may depend on the activity of the renin-angiotensin-aldosterone system, treatment with ACE inhibitors, including VASOTEC, may be associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death.

In clinical studies in hypertensive patients with unilateral or bilateral renal artery stenosis, increases in blood urea nitrogen and serum creatinine were observed in 20% of patients. These increases were almost always reversible upon discontinuation of enalapril and/or diuretic therapy. In such patients, renal function should be monitored during the first few weeks of therapy.

Some patients with hypertension or heart failure with no apparent preexisting renal vascular disease have developed increases in blood urea and serum creatinine, usually minor and transient, especially when VASOTEC has been given concomitantly with a diuretic. This is more likely to occur in patients with preexisting renal impairment. Oosage reduction and/or discontinuation of the diuretic and/or VASOTEC may be required.

Evaluation of patients with hypertension or heart failure should always include assessment of renal function. (See DOSAGE AND ADMINISTRATION.)

Hypokalemia: Elevated serum potassium ($>5.7 \text{ mEq/L}$) was observed in approximately 1% of hypertensive patients in clinical trials. In most cases these were isolated values which resolved despite continued therapy. Hyperkalemia was a cause of discontinuation of therapy in 0.28% of hypertensive patients. In clinical trials in heart failure, hyperkalemia was observed in 3.8% of patients, but was not a cause for discontinuation.

Risk factors for the development of hyperkalemia include renal insufficiency, diabetes mellitus, and the concomitant use of potassium-sparing diuretics, potassium supplements, and/or potassium-containing salt substitutes, which should be used cautiously, if at all, with VASOTEC. (See Drug Interactions.)

Surgery/Anesthesia: In patients undergoing major surgery or during anesthesia with agents that produce hypotension, enalapril may block angiotensin II formation secondary to compensatory renin release. If hypotension occurs and is considered to be due to this mechanism, it can be corrected by volume expansion.

Information for Patients:

Angioedema: Angioedema, including laryngeal edema, may occur especially following the first dose of enalapril. Patients should be so advised and told to report immediately any signs or symptoms suggesting angioedema (swelling of face, extremities, eyes, lips, tongue, difficulty in swallowing or breathing) and to take no more drug until they have consulted with the prescribing physician.

Hypotension: Patients should be cautioned to report lightheadedness, especially during the first few days of therapy. If actual syncope occurs, the patients should be told to discontinue the drug until they have consulted with the prescribing physician.

All patients should be cautioned that excessive perspiration and dehydration may lead to an excessive fall in blood pressure because of reduced fluid volume. Other causes of volume depletion such as vomiting or diarrhea may also lead to a fall in blood pressure; patients should be advised to consult with the physician.

Hyperkalemia: Patients should be told not to use salt substitutes containing potassium without consulting their physician.

Neutropenia: Patients should be told to report promptly any indication of infection (e.g., sore throat, fever) which may be a sign of neutropenia.

NOTE: As with many other drugs, certain advice to patients being treated with enalapril is warranted. This information is intended to aid in the safe and effective use of this medication. It is not a disclosure of all possible adverse or intended effects.

Drug Interactions:

Hypotension. Patients on Diuretic Therapy: Patients on diuretics and especially those in whom diuretic therapy was recently instituted may occasionally experience an excessive reduction of blood pressure after initiation of therapy with enalapril. The possibility of hypotensive effects with enalapril can be minimized by either discontinuing the diuretic or increasing the salt intake prior to initiation of treatment with enalapril. If it is necessary to continue the diuretic, provide close medical supervision after the initial dose for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and DOSAGE AND ADMINISTRATION.)

Agents Causing Renin Release: The antihypertensive effect of VASOTEC is augmented by antihypertensive agents that cause renin release (e.g., diuretics).

Other Cardiovascular Agents: VASOTEC has been used concomitantly with beta-adrenergic-blocking agents, methyl-dopa, nitrates, calcium-blocking agents, hydralazine, prazosin, and digoxin without evidence of clinically significant adverse interactions.

Agents Increasing Serum Potassium: VASOTEC attenuates potassium loss caused by thiazide-type diuretics. Potassium-sparing diuretics (e.g., spironolactone, triamterene, or amiloride), potassium supplements, or potassium-containing salt substitutes may lead to significant increases in serum potassium. Therefore, if concomitant use of these agents is indicated because of demonstrated hypokalemia, they should be used with caution and with frequent monitoring of serum potassium. Potassium-sparing agents should generally not be used in patients with heart failure receiving VASOTEC.

Lithium: Lithium toxicity has been reported in patients receiving lithium concomitantly with drugs which cause elimination of sodium, including ACE inhibitors. A few cases of lithium toxicity have been reported in patients receiving concomitant VASOTEC and lithium and were reversible upon discontinuation of both drugs. It is recommended that serum lithium levels be monitored frequently if enalapril is administered concomitantly with lithium.

Proprietary Category C: There was no fetotoxicity or teratogenicity in rats treated with up to 200 mg/kg/day of enalapril (333 times the maximum human dose). Fetalotoxicity, expressed as a decrease in average fetal weight, occurred in rats given 1200 mg/kg/day of enalapril but did not occur when these animals were supplemented with saline. Enalapril was not teratogenic in rabbits. However, maternal and fetal toxicity occurred in some rabbits at doses of 1 mg/kg/day or more. Saline supplementation prevented the maternal and fetal toxicity seen at doses of 3 and 10 mg/kg/day, but not at 30 mg/kg/day (50 times the maximum human dose).

Radioactivity was found to cross the placenta following administration of labeled enalapril to pregnant hamsters. There are no adequate and well-controlled studies of enalapril in pregnant women. However, data are available that show enalapril crosses the human placenta. Because the risk of fetal toxicity with the use of ACE inhibitors has not

been clearly defined, VASOTEC® (Enalapril Maleate, MSD) should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Postmarketing experience with all ACE inhibitors thus far suggests the following with regard to pregnancy outcome. Inadvertent exposure limited to the first trimester of pregnancy has not been reported to affect fetal outcome adversely. Fetal exposure during the second and third trimesters of pregnancy has been associated with fetal and neonatal morbidity and mortality.

When ACE inhibitors are used during the later stages of pregnancy, there have been reports of hypotension and decreased renal perfusion in the newborn. Oligohydramnios in the mother has also been reported, presumably representing decreased renal function in the fetus. Infants exposed *in utero* to ACE inhibitors should be closely observed for hypotension, oliguria, and hyperkalemia. If oliguria occurs, attention should be directed toward support of blood pressure and renal perfusion with the administration of fluids and pressors as appropriate. Problems associated with prematurity such as patent ductus arteriosus have occurred in association with maternal use of ACE inhibitors, but it is not clear whether they are related to ACE inhibition, maternal hypertension, or the underlying prematurity.

Nursing Mothers: Milk in lactating rats contains radioactivity following administration of ^{14}C enalapril maleate. It is not known whether this drug is secreted in human milk. Because many drugs are secreted in human milk, caution should be exercised when VASOTEC is given to a nursing mother.

Pediatric Use: Safety and effectiveness in children have not been established.

Adverse Reactions: VASOTEC has been evaluated for safety in more than 10,000 patients, including over 1000 patients treated for one year or more. VASOTEC has been found to be generally well tolerated in controlled clinical trials involving 2987 patients.

HYPERTENSION: The most frequent clinical adverse experiences in controlled trials were headache (5.2%), dizziness (4.3%), and fatigue (3%).

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in controlled clinical trials were diarrhea (1.4%), nausea (1.4%), rash (1.4%), cough (1.3%), orthostatic effects (1.2%), and asthenia (1%).

HEART FAILURE: The most frequent clinical adverse experiences in both controlled and uncontrolled trials were dizziness (7.9%), hypotension (6.7%), orthostatic effects (2.2%), syncope (2.2%), cough (2.2%), chest pain (2.1%), and diarrhea (2.1%).

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in both controlled and uncontrolled clinical trials were fatigue (1.8%), headache (1.8%), abdominal pain (1.6%), asthenia (1.6%), orthostatic hypotension (1.6%), vertigo (1.6%), angina pectoris (1.5%), nausea (1.3%), vomiting (1.3%), bronchitis (1.3%), dyspnea (1.3%), urinary tract infection (1.3%), rash (1.3%), and myocardial infarction (1.2%).

Other serious clinical adverse experiences occurring since the drug was marketed or adverse experiences occurring in 0.5% to 1% of patients with hypertension or heart failure in clinical trials in order of decreasing severity within each category.

Cardiovascular: Cardiac arrest, myocardial infarction or cerebrovascular accident, possibly secondary to excessive hypertension in high-risk patients (see WARNINGS, Hypotension); pulmonary embolism and infarction; pulmonary edema, rhythm disturbances, atrial fibrillation, palpitation

Digestive: Ileus, pancreatitis, hepatitis (hepatocellular or cholestatic jaundice), melena, anorexia, dyspepsia, constipation, glossitis, stomatitis, dry mouth.

Musculoskeletal: Muscle cramps

Nervous/Psychiatric: Depression, confusion, ataxia, somnolence, insomnia, nervousness, paresthesia

Urogenital: Renal failure, oliguria, renal dysfunction (see PRECAUTIONS and OOSAGE AND ADMINISTRATION).

Respiratory: Bronchospasm, rhinorrhea, sore throat and hoarseness, asthma, upper respiratory infection.

Skin: Exfoliative dermatitis, toxic epidermal necrolysis, Stevens-Johnson syndrome, herpes zoster, erythema multiforme, urticaria, pruritis, alopecia, flushing, hyperhidrosis

Special Senses: Blurred vision, taste alteration, anosmia, tinnitus, conjunctivitis, dry eyes, tearing. A symptom complex has been reported which may include a positive ANA, an elevated erythrocyte sedimentation rate, arthralgias/arthritis, myalgias, fever, serositis, vasculitis, leukocytosis, eosinophilia, photosensitivity, rash, and other dermatologic manifestations.

Angioedema: Angioedema has been reported in patients receiving VASOTEC (0.2%). Angioedema associated with laryngeal edema may be fatal. If angioedema of the face, extremities, lips, tongue, glottis, and/or larynx occurs, treatment with VASOTEC should be discontinued and appropriate therapy instituted immediately. (See WARNINGS.)

Hypotension: In the hypertensive patients, hypotension occurred in 0.9% and syncope occurred in 0.5% of patients following the initial dose or during extended therapy. Hypotension or syncope was a cause for discontinuation of therapy in 0.1% of hypertensive patients. In heart failure patients, hypotension occurred in 6.7% and syncope occurred in 2.2% of patients. Hypotension or syncope was a cause for discontinuation of therapy in 1.9% of patients with heart failure (see WARNINGS.)

Clinical Laboratory Test Findings

Serum Electrolytes: Hyperkalemia (see PRECAUTIONS), hyponatremia

Creatinine, Blood Urea Nitrogen: In controlled clinical trials, minor increases in blood urea nitrogen and serum creatinine, reversible upon discontinuation of therapy, were observed in about 0.2% of patients with essential hypertension treated with VASOTEC alone. Increases are more likely to occur in patients receiving concomitant diuretics or in patients with renal artery stenosis. (See PRECAUTIONS.) In patients with heart failure who were also receiving diuretics with or without digitalis, increases in blood urea nitrogen or serum creatinine, usually reversible upon discontinuation of VASOTEC and/or other concomitant diuretic therapy, were observed in about 11% of patients. Increases in blood urea nitrogen or creatinine were a cause for discontinuation in 1.2% of patients.

Hemoglobin and Hematocrit: Small decreases in hemoglobin and hematocrit (mean decreases of approximately 0.3 g/dL and 1.0 vol.%, respectively) occur frequently in either hypertension or heart failure patients treated with VASOTEC but are rarely of clinical importance unless another cause of anemia coexists. In clinical trials, less than 0.1% of patients discontinued therapy due to anemia.

Other (Causal Relationship Unknown): In marketing experience, rare cases of neutropenia, thrombocytopenia, and bone marrow depression have been reported. A few cases of hemolysis have been reported in patients with G6PD deficiency.

Liver Function Tests: Elevations of liver enzymes and/or serum bilirubin have occurred.

Dosage and Administration: **Hypertension:** In patients who are currently being treated with a diuretic, symptomatic hypotension occasionally may occur following the initial dose of VASOTEC. The diuretic should, if possible, be discontinued for two to three days before beginning therapy with VASOTEC to reduce the likelihood of hypotension. (See WARNINGS.) If the patient's blood pressure is not controlled with VASOTEC alone, diuretic therapy may be resumed. If the diuretic cannot be discontinued, an initial dose of 2.5 mg should be used under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and PRECAUTIONS, Drug Interactions.)

The recommended initial dose in patients not on diuretics is 5 mg once a day. Dosage should be adjusted according to blood pressure response. The usual dosage range is 10 to 40 mg per day administered in a single dose or in two divided doses. In some patients treated once daily, the antihypertensive effect may diminish toward the end of the dosing interval. In such patients, an increase in dosage or twice-daily administration should be considered. If blood pressure is not controlled with VASOTEC alone, a diuretic may be added.

Concomitant administration of VASOTEC with potassium supplements, potassium salt substitutes, or potassium-sparing diuretics may lead to increases of serum potassium (see PRECAUTIONS).

Dosage Adjustment in Hypertensive Patients with Renal Impairment: The usual dose of enalapril is recommended for patients with a creatinine clearance $\leq 30 \text{ mL/min}$ (serum creatinine of up to approximately 3 mg/dL). For patients with creatinine clearance $\leq 30 \text{ mL/min}$ (serum creatinine $\geq 3 \text{ mg/dL}$), the first dose is 2.5 mg once daily. The dosage may be titrated upward until blood pressure is controlled or to a maximum of 40 mg daily.

Heart Failure: VASOTEC is indicated as adjunctive therapy with diuretics and digitalis. The recommended starting dose is 2.5 mg once or twice daily. After the initial dose of VASOTEC, the patient should be observed under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and PRECAUTIONS, Drug Interactions.) If possible, the dose of the diuretic should be reduced, which may diminish the likelihood of hypotension. The appearance of hypotension after the initial dose of VASOTEC does not preclude subsequent careful dose titration with the drug, following effective management of the hypotension. The usual therapeutic dosing range for the treatment of heart failure is 5 to 20 mg daily given in two divided doses. The maximum daily dose is 40 mg. Once-daily dosing has been effective in a controlled study, but nearly all patients in this study were given 40 mg, the maximum recommended daily dose, and there has been much more experience with twice-daily dosing. In addition, in a placebo-controlled study which demonstrated reduced mortality in patients with severe heart failure (NYHA Class IV), patients were treated with 2.5 to 40 mg per day of VASOTEC, almost always administered in two divided doses. (See CLINICAL PHARMACOLOGY, Pharmacodynamics and Clinical Effects.) Dosage may be adjusted depending upon clinical or hemodynamic response (see PRECAUTIONS).

Dosage Adjustment in Patients with Heart Failure and Renal Impairment or Hyponatremia: In patients with heart failure who have hyponatremia (serum sodium $< 130 \text{ mEq/L}$) or with serum creatinine $> 1 \text{ mg/dL}$, therapy should be initiated at 2.5 mg daily under close medical supervision. (See OOSAGE AND ADMINISTRATION, Heart Failure, WARNINGS, and PRECAUTIONS, Drug Interactions.) The dose may be increased to 2.5 mg b.i.d., then 5 mg b.i.d., and higher as needed, usually at intervals of four days or more, if at the time of dosage adjustment there is not excessive hypotension or significant deterioration of renal function. The maximum daily dose is 40 mg.

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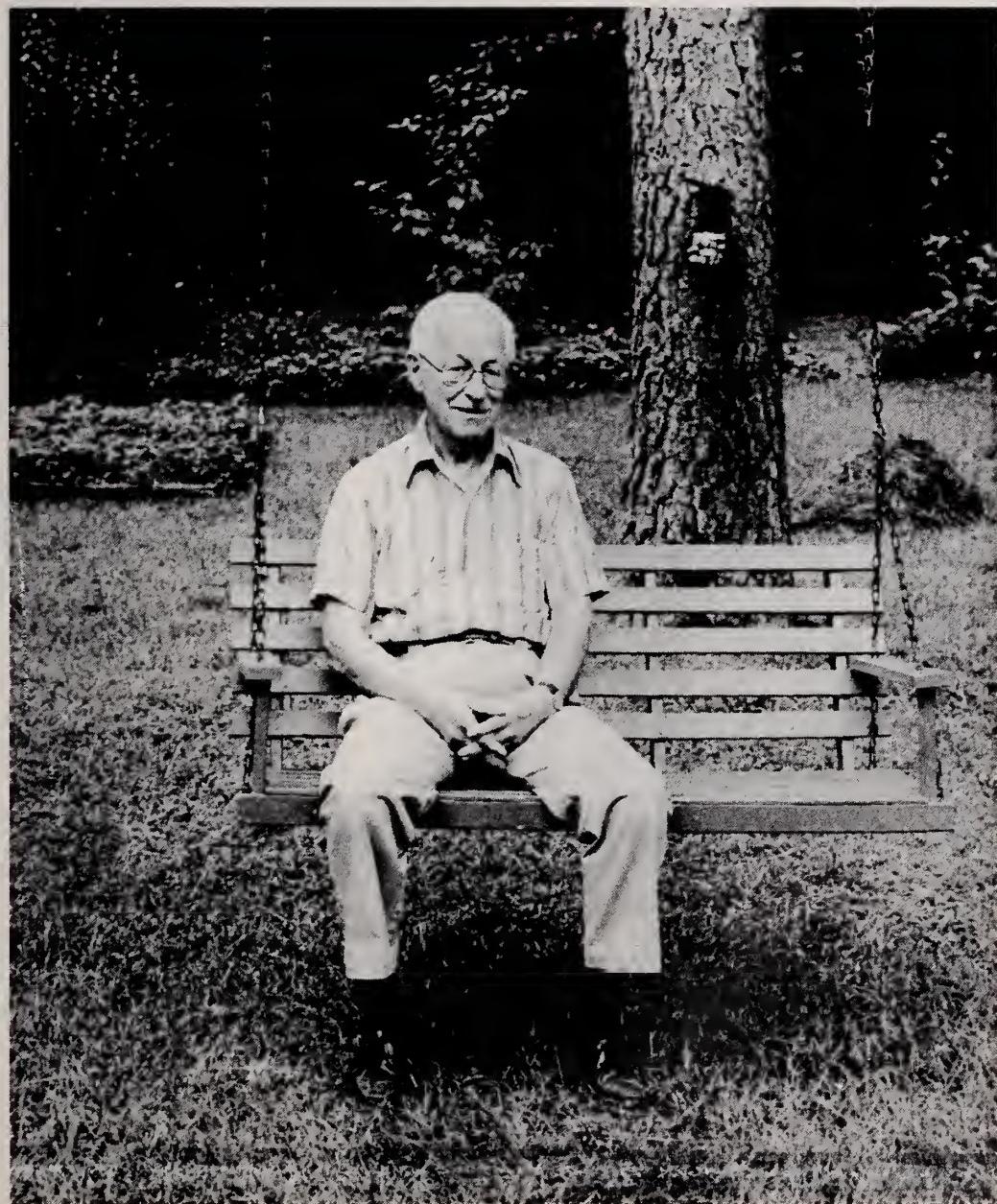
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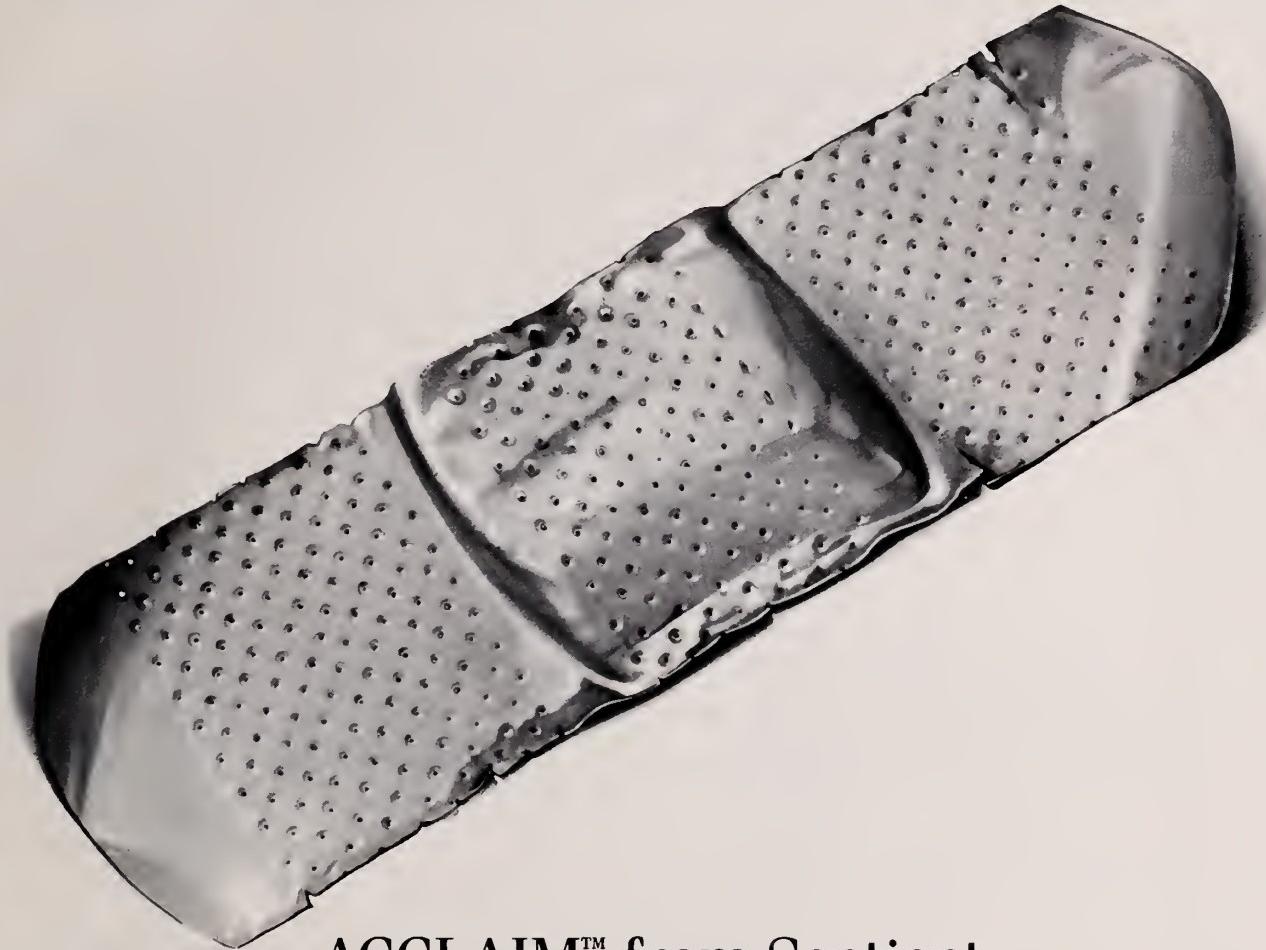
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LETTERS

Another critic heard from

Dr. Bickers criticises VIRGINIA MEDICAL for being "dull, flat" and having "no wit."¹ I would go further and question the integrity of its editorial policies.

I was asked to review *Hidden Illness in the White House* by Crispell and Gomez for VIRGINIA MEDICAL. I submitted a 2,000-word review in which I said that it was an unscholarly work derived almost solely from secondary sources, some of which were misquoted; my review was deemed unacceptable. Subsequently a 700-word review of the book by a lawyer appeared in VIRGINIA MEDICAL.² I then wrote a letter to the Editor pointing out that the printed review was "confined to the legal aspects of presidential disability described in the last chapter of the book. The bulk of the book, which deals with the medical histories of Woodrow Wilson, Franklin D. Roosevelt, and John F. Kennedy, is ignored." I described the book as "a rewrite of already published material" and went on to say that "This near-plagiarism reaches its height in four illustrations copied from the Blairs' book, from which not

only the pictures but also the captions are reproduced!" My letter was returned. The readers of VIRGINIA MEDICAL were therefore given a limited, even biased, description of the book.

As a former editor of a medical journal, I respect the right of an editor to reject any material submitted for publication. I cannot accept, however, what appears to be a deliberate circumvention of truthful, and verifiable, facts in favour of watered-down comments, presumably to avoid an unpleasant confrontation with a book's authors.

F. J. Spencer, MD, MPH

560 Caroline Drive
Ruther Glen, VA 22546

1. Bickers WM. Letters. Va Med 1990;117:6-7

2. Grad JD. Books. Va Med 1989;116:151

The Editor replies: Since book reviews are not scientific material, but rather opinion/point of view, the Editors reserve the right to approve such reviews before publication. Perhaps the journal of which Dr. Spencer is a former editor would publish his review.

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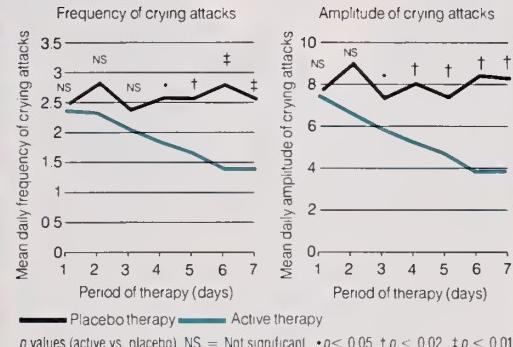
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1. Kanwaljit SS, Jasbir KS. Simethicone in the management of infant colic. *Practitioner* 1988;232:508

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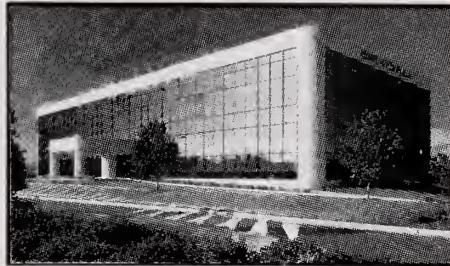
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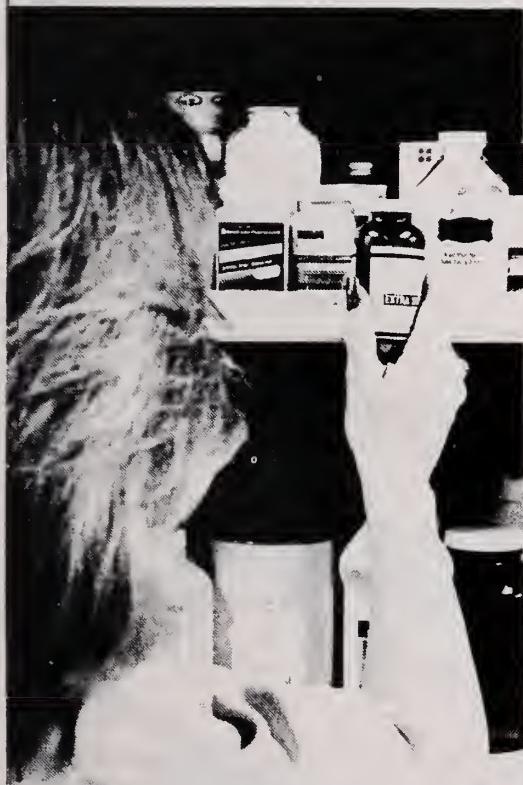
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June 4-8

General Surgery Seminar: Update and Review (Lloyd Noland Hospital), *Hilton Head Island, South Carolina*. CME Office, 205-783-5276.

June 6-10

7th Annual Meeting of the Southern Orthopaedic Association, *Maui, Hawaii*. Kathy McLendon, 205-945-1848.

June 7-9

Update in Obstetrics (American College of Obstetricians/Gynecologists), *Norfolk*. 16 credit hrs. Fee, \$515. ACOG Registrar, 800-673-8444.

June 7-10

14th Annual Postgraduate Course on Rehabilitation of the Brain-Injured Adult and Child (Medical College of Virginia/VCU), *Williamsburg*. CME Office, 804-786-7290.

June 11-15

Adult Infectious Disease Seminar: Current Update (Lloyd Noland Hospital), *Hilton Head Island, South Carolina*. CME Office, 205-783-5276.

June 14-16

35th Annual Great Smokey Mountains Pediatric Seminar (University of Tennessee), *Gatlinburg, Tennessee*. Abby Hall, 615-544-9190.

June 15-16

Arrhythmias: Interpretation, Diagnosis and Management (Medical Education Resources), *Williamsburg*. Deborah Helton, 800-421-3756.

June 20-23

Second Baltimore Perinatal Colloquium (Johns Hopkins/University of Maryland), *Baltimore*. 24 credit hrs. Office of CME, 301-955-2959.

June 21-24

Annual Meeting, Virginia Society of Ophthalmology, *Norfolk*. Donna Scott, 804-353-2721.

June 22-24

Clinical Pediatrics (American College of Pediatrics), *Washington, DC*. 16 credit hrs. CME Registration, 800-421-0589.

June 24-27

17th Annual Harvard Medical School Course on Interstate Care Medicine: The Art and Science of Critical Care, *Boston Massachusetts*. Bart Chernow, MD, 617-726-2858.

June 26-30

Family Practice Seminar: Update and Review (Lloyd Noland Hospital), *Hilton Head Island, South Carolina*. CME Office, 205-783-5276.

July 16-20

Control of Biohazards in the Research Laboratory (Johns Hopkins), *Baltimore*. Byron S. Tepper, PhD, 301-955-5918, or Dr. Jacqueline Corn, 301-955-2609.

July 20-22

Practical Internal Medicine (Medical College of Virginia/VCU), *Virginia Beach*. CME Office, 804-786-0494.

July 26-29

40th Annual Scientific Assembly of the Virginia Academy of Family Physicians, *Cavalier Hotel, Virginia Beach*. 21½ credit hrs. VAFP headquarters, 804-358-1721.

July 27-29

12th Annual Pediatric Primary Care Conference: Pediatrics at the Beach (Medical College of Virginia/VCU), *Virginia Beach*. CME office, 804-786-0494.

September 8

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How to Sell a Solo Practice

Planning for the sale of a practice should begin two years before you intend to leave, say the authors.

They offer guidelines to setting a price and on negotiating that price with a prospective buyer.

A physician who has put blood, sweat and tears into a practice expects some return when finally the decision is made to sell it. To receive that full value, however, there needs to be careful planning and preparation. Realistically, you wouldn't expect to buy a new home without getting the facts; don't expect someone to buy your practice without knowing all the important details as well. Much the same as in selling a house, selling a practice takes time. Expect to spend at least six months to one year searching for the appropriate buyer, negotiating the terms and finalizing the deal. And also don't forget the transition period, for the most crucial step of a practice transfer is the period in which the buyer and seller work together and cement their relationship. This period can easily extend to three or four months.

Planning is essential, and our gen-

eral advice is to begin at least two years before you want to leave your practice. Chiefly, your planning must deal with the following three questions.

Do you have something to sell? Take a good hard look at your practice. Is the gross revenue healthy? Hopefully, your income hasn't dropped and your work levels in past years are consistent. How is the location? Would someone want to spend most of their time where you are currently practicing? If the demographics of the neighborhood

have changed since you first hung out your shingle, incorporate that into your thinking.

How is the competition? Moderate competition is a good thing; no competition usually means there is no reason to buy. Why? Either the area is considered less than desirable and is underserved, or the population can't support another practitioner. In the first scenario, a candidate could set up down the street and have a healthy income without buying; in the second, why would he buy when you're leaving anyway? If your area needs only one physician and you're going, the candidate can wait until you're gone, and set up shop.

Keep your plans to yourself and your advisors. The biggest mistake you can make is to let your best friend (who's also your best referral) know that you are moving to Florida as soon as you sell the practice or by

This text was excerpted from a series of articles developed by The Health Care Group, a practice management consulting firm © 1990. The principal authors were Mark E. Kropiewnicki, JD, LLM, and Dorothy R. Sweeney of The Health Care Group, Meetinghouse Business Center, 140 West Germantown Pike, Suite 200, Plymouth Meeting PA 19462.

January 1, whichever comes first. The word *does* get out and all of your potential buyers will either vanish until December 15 or try and push the price down. When you are interviewing prospective candidates, be honest and straight-forward about anything they'll need to know, but they don't need to know how anxious you are to start your retirement!

How much? If you want an objective view of what your practice is worth, have it appraised. Besides analyzing your location, competition and your revenue and expenses, a good appraisal will give you an idea of what the market can handle. Unfortunately, there is no magic formula you can apply to come up with the "right" selling price. In fact, there are many valuation techniques, and depending on what journal you're reading or which accountant you're talking to, there are a dozen "standard" methods to value a medical practice. No single method has proven to be correct or accepted in all cases. The "real" value will be determined when the deal is signed. And it is usually one area where everyone has an opinion.

However, the dollar figure given by an appraiser is a good starting point. Be sure that whoever puts a value on your practice has previous experience with medical practice sales; don't rely on the accountant or attorney who is taking this on as a "new" project.

Of all the methods that are currently being used, the Gross Revenue Multiplier is the most widely quoted. The multiplier is the ratio of practice value to annual revenues. In other words, a practice with a price tag of \$100,000 and a gross revenue of \$200,000 is selling at 50% of gross revenue. While the multiplier is helpful in establishing a benchmark in which to start negotiations, every practice has different characteristics, which will affect its value. Just because Dr. Smith's practice across town with the same specialty as yours sold for 60% of his average gross, doesn't mean that is your magic number too. When a practice comes to market, each buyer and

seller will rely on the multiplier as a starting point. But, since the multiplier represents an average of practices evaluated, it should not be quoted as the ultimate authority.

Many sellers (regardless of specialty, years in existence, location or profitability) tell us they expect to receive one year's gross income for the sale of their practice. It just isn't so! Rarely do we ever hear of a practice selling for a number that high. Primary care practices that can offer the benefits of referrals to their neighborhood hospitals will sometimes approach that number, but more times than not that number was quoted by an advisor who wasn't aware of what the market will bear for medical practice sales.

Sellers who insist on starting their negotiations at such a high level will soon find potential buyers are scared away. The best way to justify your selling price is to find evidence of it happening before. But remember, each practice is unique and requires individual analysis for determination of fair market value. Know the market before you begin, and recognize that each person in a buy-sell transaction may feel a little dissatisfied when the deal is finished—but it is certainly better than no deal at all.

GOODWILL

In most practice sales, the goodwill factor is not readily isolated. A sale typically is negotiated for the practice as a whole, not for its component parts.

Subject to numerous variables applicable to each particular circumstance, the following are our updated goodwill guidelines.

Primary Care. These practices (family practice, general practice, pediatrics, and office-based primary care internal medicine, for example) still sell at around 50% of annual gross income, though we see a slight upward trend. In a negotiating situation, we might now suggest asking for 60% (or even slightly more), hoping to sell at a bit over 50%.

Referral Practices. Because both

general and specialty surgeons rely largely on physician referrals for most of their work, as do subspecialty internists like cardiologists and G.I. doctors, their practice goodwill values are less. Our guideline is 20% to 30% of annual gross income although some may be in the mid-30s.

Ophthalmology. Because it combines the ongoing patient volume of a primary care practice with the likelihood of surgery for some of those patients, ophthalmology is still probably the most "valuable" specialty of them all; however, we are concerned that continued fee pressures will further depress the goodwill figure. By the very nature of its practice mix, ophthalmology should have a greater economic value than primary care. There is still an active market in the sale of ophthalmology practices, partly because many doctors believe that "market share" of patients will be vital if reimbursements fall further.

Ob/Gyn. The value of this specialty is puzzling, and we estimate it at 30% to 40% of gross annual receipts. The scarcity of these specialists, due to the malpractice climate and the tough on-call demands, makes a practice sale far more doubtful than in other specialties.

Dermatology, Allergy, Neurology. These internal medicine-related practices have many economic characteristics similar to those of primary care. Dermatology is holding up to a 50% of gross guideline, but allergy seems to be slipping. An allergist may ask 50%, but should be prepared to settle for less, especially if the practice lacks a very strong patient base. Neurologists are somewhat above subspecialty internists as to goodwill value, so our guideline range is 35% to 50% of annual receipts.

RAP. The hospital-based practices, like radiology, anesthesiology, and pathology, have no pattern. Their values depend too much on hospital contracts, so that there is often no true sale likelihood at all. We assume no more than a minimal, 10% of gross valuation, merely to

recognize the practice as a going concern.

TAX CONSIDERATIONS

A significant factor to consider in arriving at the sales price will be how the sales transaction will affect income taxes of both the buyer and seller.

1. Equipment and Furnishings. From the buyer's tax standpoint, the highest possible allocation to practice equipment and furnishings is desirable. The buyer benefits from an election to immediately deduct as an expense up to \$10,000 of the hard assets' cost and from depreciation deductions, usually over five or seven years. The buyer thus can write off the purchase price of hard assets and thereby reduce their after-tax cost.

The seller will usually have less favorable tax benefits on hard assets. While some of the price may be nontaxable, as return on undepreciated original cost, most of the purchase price will probably be taxed as ordinary income—to the extent of depreciation taken in prior years. If the seller has not owned an item generally for at least five years, he/she would have to repay part of all of any investment tax credit previously taken on those assets.

2. Goodwill. Any amount paid for good will cannot be depreciated or deducted at all. Thus, the buyer will negotiate hard to keep the contract amount attributable to goodwill at a minimum.

If the seller did not purchase the practice from another physician, his/her tax basis in goodwill is zero. Thus, the entire amount attributable to goodwill will be taxed at the maximum income tax rate.

3. Accounts Receivable. Practice sales generally do not include accounts receivable. Buyers usually do not wish to pay for amounts that may or may not be collected from patients or third-party payers. In addition, Medicare and Medicaid rules prohibit the sale or assignment of

continued on page 199

Who's Who:

Dr. John A. Murray of Franklin has retired, thus winding up 36 years of pediatrics and 14 years on the City Council, two years as Franklin's mayor, and a term as president of the Franklin/Southampton Chamber of Commerce. Dropping out of such a productive life will be a big change, and he wasn't sure he was going to like it when he was interviewed by Jim Hekel of the *Tidewater News*. "The kids," that's what he'll miss most, he said. "I've always enjoyed the kids. They are refreshing and invigorating, simply amazing."

Dr. Murray was born in Richwood, West Virginia, where his father was a pharmacist. In the Navy, during World War II, he had a taste of medicine as a medical corpsman and, when he returned from the service, he taught school while waiting for admission to medical school. He graduated in 1952 from the Medical College of Virginia and the next year, after canvassing the state for possible practice locations, settled in Franklin.

City politics were a magnet for Dr. Murray. "I'm interested in the operations of the community, especially the schools," he told the reporter. "During my time on council we established the city's first industrial park, built new schools, and set a limit of two terms as a maximum anyone can serve on the School Board. I would like to see the two-term standard imposed on all public offices, from US Senator on down."

Dr. Murray and his wife Margaret have four children, all now grown and scattered to Richmond, California, DC, and New York.

For her "indelible mark on Warren County of a lifetime of service to her community," **Dr. Elizabeth B. Sherman** was named Citizen of the Year by the *Front Royal News* late

last year. Earlier, the 90-year-old physician had retired (Va Med March 1989), telling a reporter for the *News*, "I'm tired, if you want to know the truth."

Graduated at the top of her class from the University of Maryland Baltimore County Medical School in 1926, Dr. Sherman trained for general practice, including a residency in pediatrics in New York City, before opening her office in Front Royal in 1934. She was instrumental in organizing efforts to build Warren Memorial Hospital and is now on that hospital's emeritus medical staff.

After practicing dermatology in Danville together for more than 30 years, **Drs. Eugene M. Evans, Jr., and Walter C. Fitzgerald** closed their office last year and retired together.

Dr. Fitzgerald, a graduate of the University of Virginia School of Medicine (1943), was the only dermatologist in the county when he began the practice in 1953. Four years later he met Dr. Evans at Duke University, where Evans was finishing his medical studies and Fitzgerald was doing some consulting, and the two turned the practice into a partnership.

The first item on Dr. Fitzgerald's retirement agenda is to make Buggs Island his permanent home, not only for its waterside attractions but because it's a great place for runners. Dr. Fitzgerald began running 30 years ago and at 70 is a marathon class competitor who was the winner in his age division in a recent Richmond Marathon.

Dr. Evans intends to travel, travel, travel. He recently completed a 5,400-mile motor trip through the West and now is looking toward "a whole world out there I want to see," he told a reporter for the Danville *Bee*, especially the Pacific Is-

Retirements

lands, Australia, and Japan.

It took three years, the dermatologists recalled, to find a doctor to take over the pair's practice. He is **Dr. Michael C. White**, who was graduated from the Medical College of Virginia/VCU in 1981 and used to call Norfolk his home.

A medical career spanning both private practice and public health ended last year when **Dr. John M. Stirewalt** of Waynesboro retired from Western State Hospital. He had served his entire professional career in Augusta County and was at Western State for 22 years.

Both in New Market, Virginia, Dr. Stirewalt was graduated from both the University of Virginia and its School of Medicine (Class of 1951) and trained in ENT at the university's hospital. He first entered public health as medical director for Woodrow Wilson Rehabilitation Center, Fishersville, then changed to a private practice in Waynesboro, returning to public health as assistant director for the Augusta/Staunton Health District. At Western State, which he joined in 1966, Dr. Stirewalt filled both clinical and administrative posts, including chief of staff of the medical center.

Throughout his career, Waynesboro has been home base for Dr. Stirewalt and his wife, the former Virginia Dean. They have four children and four grandchildren.

After 31 years of practice in Alleghany County, **Dr. Alvin W. Finestone** has retired from the radiology department of Alleghany Regional Hospital, Low Moor. He had been at Alleghany Regional since it opened ten years ago; before that he was at the Chesapeake & Ohio Hospital

(now Emmett Memorial) in Clifton Forge.

Born in Philadelphia, Dr. Finestone earned his MD in medicine in his hometown at Temple University's School of Medicine, then trained at hospitals in New Jersey, New York, and Pennsylvania. Looking for a practice site after completing a tour of duty with the Army, Dr. Finestone answered an ad in *JAMA* seeking a small-town radiologist and soon was on his way with wife Elaine to Clifton Forge. He joined **Dr. George N. Chucker**, now also retired, at Chesapeake & Ohio, and they were that hospital's entire radiology department from 1958-1970.

Dr. and Mrs. Finestone have reared three children; one of them, **Douglas H. Finestone, MD**, followed his father into medicine and is now practicing psychiatry in Greenville, North Carolina. His father has not given up radiology entirely; to keep his hand in, as he puts it, he is working 10-12 weeks a year for Comp Health, a national firm which he represents at exhibits around the country.

As for his erstwhile associate, Dr. Chucker, who retired in 1986, he was notified recently that he has been elected a fellow emeritus of the American College of Radiology. A past president of the Virginia Chapter, ACR, Dr. Chucker was for six years a member of the national organization's Board of Councilors.

In Urbanna, **Dr. Arthur L. Van Name, Jr.**, Medical College of Virginia '36, past president of the Mid-Tidewater Medical Society, and present member of the State Board of Medicine, has retired after 40 years of general practice, but he doesn't want anything said about it, so we won't.

When **Dr. Jorge A. Naranjo** of Boykins retired late last year after 25 years of family practice, Mayor Rick Francis signed a proclamation honoring him as a "selfless, caring humanitarian" and declared Sunday, October 8, as Jorge A. Naranjo Appreciation Day. The community followed up with a public celebration of the Day at the Boykins firehouse.

A native of Ecuador, Dr. Naranjo came to the United States to study medicine, and, after a residency at Louise Obici Memorial Hospital in Suffolk, established his private practice in Boykins in 1964.

In Culpeper, the community's first full-time surgeon is retiring after nearly 30 years of practice at Culpeper Memorial Hospital. He is **Dr. Grahame F.T.W. Henson**, a Welchman who came to the States after practice in a London hospital during the air raids of World War II and stints with the British Army in India and British Somalia. He was on the junior attending staff at the University of Virginia when Culpeper Memorial opened its doors and asked him to come on in as its first surgeon. He started on July 4, 1960, and early on did orthopedic, gynecologic and ENT procedures, though that agenda contracted as the specialties came along. He has been a trustee on both hospital and parent board and has twice served as president of the CMH medical staff.

To mark Dr. Henson's retirement, the hospital sponsored a dinner and "roast" in his honor @ \$25 a head and designated the proceeds to the Windmore Foundation, a local art and drama organization in which the surgeon is much interested. Dramatics have long been an enthusiasm of his, perhaps, he suggested to Kathleen Hoffman of the *Culpeper News*, because "surgery and the theater

Who's Who:

have flamboyance in common." While in Africa in the forties he acted with the Nairobi Repertory Company; he headed up Culpeper's bicentennial pageant ("a big hit," according to the review); and he has usually done a star turn in the Pink Ladies' Follies, the annual hospital auxiliary benefit. He has no intention of retiring from his activities behind the footlights and in the wings, and he hopes as well to write two books, one about medicine with an autobiographical basis and the other probably a "murder thing with a hospital setting."

He was the first full-time anesthesiologist in Waynesboro. Now, after nearly 30 years of "passing the gas" at Waynesboro Community Hospital, **Dr. John P. Heatwole** has retired. But only from anesthesiology, he emphasizes. He'll go right on with a heavy schedule of projects as woodworker, church volunteer, photographer, computer buff, and writer. "I'm hoping to publish a genealogy newsletter," he told reporter Louis Andrews of the Waynesboro *News-Virginian* as he showed him a copy of "David Heatwole and His Descendants," a family history of his authorship.

After graduating in '54 from the Medical College of Virginia, Dr. Heatwole started out as a family physician in Harrisonburg, but he became interested in anesthesia and got his training for it in the Navy. In 1960 he and his wife Anita, a registered nurse, settled in Waynesboro; there they have reared two daughters and a son, all now grown. The son, **Dr. Kenneth M. Heatwole**, followed his father into medicine, graduating from MCV in 1984, and now is a pulmonary specialist at the University of Virginia Health Sciences Center in Charlottesville.

Two GPs who retired recently after long service in South Hill were honored when the Community Memorial Healthcenter gave its 1989 board of trustees/medical staff Christmas party. In the spotlight were **Dr. Thomas A. Saunders**, 39 years of service, and **Dr. Z. Dean Sadighian**, 21 years.

Dr. Charles H. Crowder, Jr., and **Dr. Wallace J. Horne** spoke in tribute to their colleague, Dr. Sadighian, who was unable to be at the ceremony. Dr. Saunders was on hand, however, to hear **Dr. F.C. Sturmer** read the resolution in praise and hand him a framed copy of it.

Each year the Women's Auxiliary of the Northampton-Accomack Memorial Hospital selects as honorary chairman of its Charity Ball a person whom it wishes to recognize for contributions to the Eastern Shore and its hospital. The most recent recipient of this honor is **Dr. E. Milton Kellam** of Belle Haven, whose selection happened to occur shortly after his retirement from surgery after almost 40 years of practice.

Born on the Eastern Shore in Belle Haven in the house where he still lives, Dr. Kellam is the son of the late **Dr. Sydney S. Kellam**, a family doctor who practiced out of his home until his death in 1933. The son went to Hampden-Sydney, then the University of Virginia School of Medicine (Class of '43), then completed his training in New Britain, Connecticut, first in pathology, then in surgery. He returned to the Eastern Shore in 1951 and went into partnership with three other surgeons. At the time the four were the only surgeons on the Eastern Shore; today there are 11 surgeons, many of them specialists.

In 1954 Dr. Kellam met and married a young woman named Joan

Lansford who was working in the hospital record room; they have three children, all now grown.

No sooner had **Dr. James E. Chapman**, Falls Church, retired from private practice and the medical directorship of Dominion Hospital than he picked up an appointment to the board of governors of the Northern Virginia Institute for Continuing Medical Education. The internist also will continue as medical advisor at a high-tech firm involved in developing an eye-operated computer system for severely disabled persons. Dr. Chapman is a graduate of George Washington University School of Medicine, Class of '49.

For 45 years **Dr. A. W. Graves V** saw patients in the big white house in Lacey Spring where his father had long practiced medicine. Then, in 1985, the general practitioner discovered he had Parkinson's disease, and that spelled retirement. He's coping nicely, according to Jim Hankey of the Harrisonburg *News-Record*, thanks to the help of his wife Ida, who was trained as a nurse, and two sons, Owen and A.W. VI, who take turns driving from Harrisonburg to Lacey Spring to accompany their father when he drives a car. Dr. Graves is a graduate of the University of Virginia School of Medicine, Class of '39, and trained at Norfolk General.

Way back in June 1989, Va Med reported that **Dr. E. M. Henderson** had closed his office in Nassawadox on the Eastern Shore. To the Editors' regret, the report was misleading. True, Dr. Henderson resigned from the professional partnership with which he had been associated, but he continues in general practice, albeit, he says, "in a limited way."

Retirements

After six decades of service to the people of Halifax County, the doors of Little Retreat Clinic in Clover were closed last September and **Dr. William J. Hagood, Jr.**, said good-bye to the patients. He had done his best to find a physician to join him in the clinic after the cousin with whom he had shared the clinic for 35 years, **Dr. Warren C. Hagood**, retired earlier in the year, but though several prospects had taken a look, none had signed on, and at 72, "Dr. Bill" Hagood couldn't go it alone.

Dr. Bill's uncle, **Dr. James D. Hagood**, pulled his nephew into the clinic in 1946 when the younger doctor got out of the Army. Dr. Jim had bought the clinic building in the early twenties from **Dr. Rawley A. Fuller**, who used it as a hospital for his surgical practice. (Dr. Fuller then moved ten miles southwest to South Boston and founded a hospital that eventually became Halifax-South Boston Community Hospital, where a son, **Dr. W. Allen Fuller**, now practices surgery.)

In 1954 Dr. Warren joined the

two, and during the following years the three doctors treated 30-45 patients a day.

Thanks to the untiring clinical support of his two associates, Dr. Jim Hagood could take the time to serve in the Virginia State Senate for 29 years (1942-1971) and in the fifties was President of the Medical Society of Virginia. He died in 1972.

Dr. Bill Hagood was MSV President, too, in 1978-79, and is currently an MSV delegate to the American Medical Association.

Five retired physicians who made significant contributions to medical service in Smyth County were recognized recently by the medical staff of Smyth County Community Hospital in Marion. They are **Dr. Charles G. Thompson**, **Dr. E. V. Richardson, Sr.**, **Dr. C. C. Hatfield**, **Dr. Joseph J. Eller**, and **Dr. J. Stuart Staley**. They were presented with plaques signifying their election to honorary membership on the hospital's medical staff, the first time such recognition has been accorded. □



Dr. Bill Hagood at the Little Retreat Clinic in 1949.

accounts receivable of Medicare and Medicaid patients.

However, for those occasions where accounts receivable are sold, that sum must be reported as ordinary income by the selling physician. The buying physician is not taxed on collection of such purchased receivables to the extent of their purchase price. A tax deduction is allowed if the buyer collects less than was paid for the accounts receivable. If more is collected on the accounts receivable than was paid for them, such excess is taxable to the buyer as ordinary income.

If accounts receivable are not sold with the practice, both parties will typically find it valuable for the purchaser to bill and collect receivables for the selling physician. Thus, practice continuity is established in patients' minds and is of benefit to the purchasing physician. In this way, the selling physician has a better chance of collecting his or her receivables and need not maintain a staff or billing system.

4. Leasehold Improvements. If there are leasehold improvements sold with the practice, the buyer will be able to depreciate the cost of those improvements on his or her tax return. Tax rules now generally require such improvements to be tax-deducted over a rather long time. The selling doctor will be taxed on any gain over the cost of those improvements.

5. Office and Equipment Leases. Occasionally, the buyer will pay the seller a premium to assume a favorable current office lease or to assume the lease on equipment that the practice rents. The amount realized by the seller is taxable. The amount paid to the buyer is depreciable, although tax rules may require these items to be depreciated over a period in excess of the remaining lease term.

6. Supplies. Supplies sold with the practice will be taxed at ordinary income rates on the amount received if supply costs were previously deducted by the practice. The buyer will be able to deduct costs of those supplies as if they were purchased in

CLASSIFIED INFORMATION

Three Virginia physicians who are looking toward retirement offer practice opportunities in the Classified Ad section of this issue of Va Med:

- "For Sale—Solo family practice in Northern Virginia"
 - "Norfolk—Retiring from 25-year solo practice of general surgery"
 - "Family practice on Chesapeake Bay, owner retiring"
- See page 222 for details.

the normal course of business.

7. Medical Charts and Records. While this is not absolutely certain, purchased patient medical charts and records *might* be treated as tangible property subject to depreciation. Useful lives of five to ten years have been claimed in many practice sales, and there are tax cases supporting such a write-off. The buyer would thus be able to deduct the portion of the purchase price allocated to medical charts and records, thereby reducing his/her after-tax cost. The seller usually has no basis in the medical charts and records, and therefore, the entire amount allocated to them will be taxable income to the seller.

8. Consulting Pay/Conovenant Not to Compete. Carefully stated and documented allocations to the selling doctor's promise to provide consultative services and/or not to compete are deductible as paid by the buying doctor. The seller is taxed upon receipt on the amounts so allocated. This has been and should continue to be a popular tax-saving approach if properly structured.

One year's financial statements; three to five years' tax returns; patient data reports (i.e., new patients, daily numbers seen, socioeconomic profile, referral sources); insurance profile (a breakdown of percentage of patients seen by each carrier); assets (including date acquired); fee schedules; practice promotional items; employee information; samples of the appointment book and day sheets; any contracts and leases; and finally, a brief history of the practice and an overview of the community.

You need to be open to a serious inquiry. This is no time to paint a picture either too rosy or too unflattering. Plan to discuss frankly hospital politics, coverage, how the competition affects your practice, and the number of hours you spend working in it each year. Hold the meeting in your office and let your serious candidates see that you have nothing to hide; allow them to observe the office systems.

THE NEGOTIATED PRICE

Once an initial sales price has been reached, continuing negotiations between the parties should concentrate on tax allocations of the total purchase price. Because there is a give-and-take between the parties in regard to tax treatments, both may be able to restate the "deal" for their benefit.

For example, the buyer's total purchase price might be increased, but the change may give him/her greater tax deductions while reducing the allocated goodwill price. The seller might correspondingly have an increase in the amount of taxable income, but come out ahead because of the higher overall purchase price. The selling physician may be in a lower tax bracket after leaving practice, so the increased price may not be taxed as heavily.

So long as the resulting overall purchase price, considering tax treatment, keeps the selling physician's after-tax yield at least the same, everyone should be pleased.

PREPARING TO MEET A BUYER

Be prepared before you meet with the buyer. If you've opted for an appraisal, have the report in hand along with the tools the appraiser used in justifying the value. The most frequently requested items are:

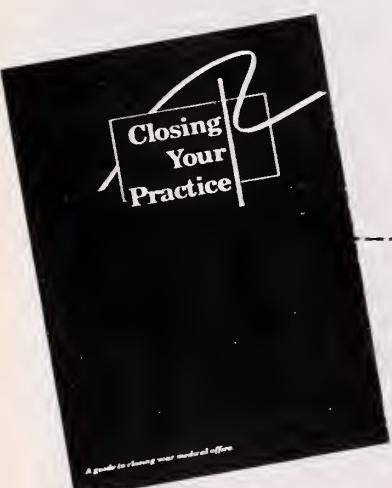
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Such restructuring may help the buyer and hence help speed the sale of the practice. Fig. 1 shows how a reallocation of the sales price lowered the ultimate after-tax cost to the buyer by \$15,600 and gave the seller a \$2,880 after-tax increase in spite of the sales price increase from \$100,000 to \$104,000.

Quite naturally, the Internal Revenue Service is interested in such price structuring. In particular, new tax laws now virtually require that some of the purchase price, and perhaps much of it if you are not careful, be allocated to goodwill. The new rule essentially requires that any portion of the purchase price in excess of the "true" fair market value (and not some agreed or negotiated figure) of the purchased tangible assets (primarily equipment and furnishings) is to be allocated to nondeductible goodwill.

Reasonable allocations of the purchase price among the various assets and items described above (including goodwill) ought to be defensible without considering taxes. However, if you are too greedy, the IRS may reject your allocation if you are audited. Nevertheless, if the parties reach an agreement and the revised pricing figures are not totally beyond reason, the mutually expected tax treatment should almost certainly follow.

It is quite common for the selling physician to accept a promissory note as partial payment for the final negotiated sales price. If the selling doctor is to become the "bank," then he or she should watch for an additional tax trap—imputed interest. The imputed interest rules today are quite complicated and involved. Suffice it to say that if the note the seller agrees to hold provides for no or low interest, the IRS will assume an appropriate interest rate as provided in the law and IRS regulations. The effect of this imputed interest rule is to convert part of the seller's purchase price allocation to interest income instead of gain from the sale of the various assets sold. Depending on the circumstances, the seller's after-tax yield may be reduced. The

Table 1. Sale Example.*
A. True Sale Price

	Price	After-Tax Yield To Seller	After-Tax Cost To Buyer
1. Equipment (assume cost basis = \$10,000)	\$ 15,000	\$13,600**	\$10,800
2. Goodwill	\$ 85,000	\$61,200	\$85,000
TOTAL	\$100,000	\$74,800	\$95,800

B. Revised Sale Price

	Price	After-Tax Yield To Seller	After-Tax Cost To Buyer
1. Equipment (basis = \$10,000)	\$ 20,000	\$17,200**	\$14,400
2. Covenant Not To Compete	\$ 30,000	\$21,600	\$21,600
3. Consulting Pay	\$ 20,000	\$14,400	\$14,400***
4. Charts and Records	\$ 15,000	\$10,800	\$10,800***
5. Goodwill	\$ 19,000	\$13,680	\$19,000
TOTAL	\$104,000	\$77,680	\$80,200

*Assumes both seller and buyer are in 28% tax bracket in all years. In actuality, this will likely not be true, and calculations should be made on yearly basis, using each year's likely tax bracket for each party.

**28% tax on price above basis. Assumes all is depreciation recapture.

***These tax results are uncertain.

buyer is allowed to deduct the amount imputed as interest. As a result, the buyer's after-tax cost generally should not be adversely affected by an IRS imputed interest determination. Thus, a buyer's promissory note should not fail to reflect a definite payment period and an adequate interest rate.

Insurance Protection. As a last consideration, it is imperative that your interests in the continuing practice be sufficiently guaranteed through the use of insurance (assuming you were not paid the entire purchase price for the practice). Any outstanding balance the buyer owes you should be protected through life insurance (on his or her life), possibly along with disability insurance in case of accident, illness, or injury to him or her (although requiring such disability insurance may be subject to negotiation due to its expense). You should be listed as the beneficiary. In addition, general insurance on the practice's contents should be required with you named as the loss payee. Be sure that these insurance requirements are properly included in the sales agreement. □

GOODWILL REGISTRY

For nine years The Health Care Group has provided a national clearinghouse service of goodwill valuations. Available primarily to medical practice advisors who participate by submitting actual transaction data, *The Goodwill Registry* summarizes the transactions and offers yearly updates on average values on a specialty-by-specialty basis. Your advisor may purchase the *Registry* by calling Joy Goodman at The Health Care Group, 215-828-3888.



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Acute Rheumatic Fever: Is It Returning to Virginia?

Michael B. Lenhart, MD, and
Duncan S. Owen, Jr., MD,
Richmond, Virginia

ACUTE rheumatic fever (ARF) is an inflammatory disease that may follow Group A streptococcal infections and may affect almost any organ system clinically. The twentieth century has witnessed a marked decline in the incidence of ARF in the developed countries, in both pediatric and adult populations.^{1,2} Since the mid-1980s, however, numerous case reports have been appearing and seem to herald a resurgence of the disease.³⁻⁶ Recently an adult was admitted to the Medical College of Virginia Hospitals with acute rheumatic fever.

Case Report

A 19-year-old black male was admitted with complaints of chest pain and fatigue. An electrocardiogram revealed a first degree A-V block and a transient second degree (Mobitz I) A-V block. An exercise thallium test was normal. Physical examination revealed a grade II/VI systolic ejection murmur but no evidence of heart failure. His symptoms improved with antacids, and he was discharged without a specific diagnosis. Five days later he returned, complaining of an acute onset of right hip and knee pain; this progressed to the point that the patient was unable to ambulate. He denied trauma, prior joint pain, drug allergies, skin rash, tick bite, penile discharge, or recent travel. The patient had no significant past medical history except for sickle cell trait, and he took no medications regularly.

At the time of admission, physical examination was significant for a temperature of 103.8°F and a grade II/VI systolic ejection murmur. A transient pericardial friction rub was heard. The right hip and knee were markedly decreased in range of motion, apparently because of pain, because only minimal swelling and heat was noted in the right knee. Admission chest radiograph was clear and electrocardiographic tracing revealed only sinus tachycardia. Laboratory data was

From the Division of Rheumatology, Allergy and Immunology, Department of Internal Medicine, Medical College of Virginia/Virginia Commonwealth University. Address correspondence to Dr. Owen at Box 647, MCV Station, Richmond VA 23298-0647.

Submitted 2-16-90.

significant for hemoglobin of 8.3g/dl and a WBC of 14,500/mm³.

At the end of the first hospital day, the patient complained of pain and swelling in his left hand and elbow. A rheumatology consultant elicited a history of a sore throat approximately 3-4 weeks prior to admission. Aspirin, 6 gms a day, was instituted. Over the next 48 hours several other joints became symptomatic, but by 72 hours he began to improve. Laboratory studies revealed negative rheumatoid factor, antinuclear antibodies, cold agglutinins, hepatitis panel, as well as negative cultures of the blood, urine, and synovial fluid. However, an antistreptolysin O titer was 960 units (normal less than 170), erythrocyte sedimentation rate 127mm/hr, and throat culture revealed group Aβ hemolytic streptococci. He was administered 1.2 million units of intramuscular benzathine penicillin. By hospital day 10 the patient was afebrile and ambulating without difficulty. He was discharged and followup visits were arranged.

Discussion

Acute rheumatic fever is defined by the revised Jones criteria.⁷ To make the diagnosis, one must have two major or one major and two minor criteria, plus supporting evidence of streptococcal infection.

- Major criteria: carditis, polyarthritis, chorea, subcutaneous nodules, or erythema marginatum.
- Minor criteria: previous history of rheumatic fever, elevated ESR, or prolonged PR interval.

The incidence of ARF has declined in the 20th century, with the nadir among children and teenagers occurring from 1970-1980, when the overall incidence ranged from 0.23-1.88 cases per 100,000 per year.¹ Also, the incidence decreased in the adult population to the point that mass penicillin prophylaxis for naval military recruits was discontinued in 1980.² In the mid-1980s there appears to be a resurgence of acute rheumatic fever, with individual case reports and outbreaks reported in Utah, Ohio, Pennsylvania, and military bases in San Diego and Missouri.^{2,3,5,8} (Several pediatric cases have been seen at the Medical College of Virginia Hospitals in the past two years.)

The patient presented here met one major and three minor criteria and had streptococcal pharyngitis. Polyarthritis was the most striking feature in our patient and in adults is the most common manifestation of ARF. The pattern of arthritis is variable and ranges from the classic migratory polyarthritis to one that is progressive and symmetrical in large joints.⁹ Our patient exhibited progressive arthritis that affected both large and small joints and even the temporomandibular joints. In all series reviewed (and as seen in our patient), the severity of the pain seemed disproportionate to the physical findings.^{2,9} Other forms of arthritis that may be confused with ARF are "reactive arthritis" associated with enteric pathogens, disseminated gonococcal infection, and adult Still's disease. A

feature of the arthritis associated with ARF is its well-described response to salicylates.⁹ The newer nonsteroidal antiflammatory drugs may be as good or better but there are no data regarding these drugs.

Carditis is generally an infrequent complication in adults with ARF. In a review of adult cases since 1957, the overall incidence of carditis was 33% (range 18-56) and in the recent outbreak in San Diego it was 30%.⁶ Clinically, carditis is manifested by one of the following: organic cardiac murmur not previously present, cardiomegaly, congestive heart failure, or pericarditis. Conduction disturbances do not necessarily indicate acute carditis.⁷ Our patient initially presented with a systolic ejection murmur and later developed a pericardial rub; however, echocardiography failed to reveal an effusion. Our patient had no evidence of chorea, erythema marginatum, or subcutaneous nodules. These are rare in adults and are seen generally in less than 5% of the cases.¹⁰ While not part of the revised Jones criteria, our patient presented with microcytic anemia. Despite an extensive workup, no definite etiology of the anemia could be determined. It is interesting that all ten patients in the San Diego military recruit study also had a "significant anemia."⁶

The treatment of rheumatic fever must be concerned with three separate aspects of the disease:

1. The precipitating streptococcal pharyngitis is treated conventionally with a 10-day course of oral penicillin. If compliance is a problem, a single intramuscular injection of benzathine penicillin 1.2 million units (for adults) is administered.

2. The inflammatory polyarthritis is treated with 4-8 grams per day of aspirin along with bed rest. The duration of treatment is guided by response with approximately 80% of patients responding (pain-free) by six weeks and greater than 90% responding by 12 weeks.¹ Our patient showed improvement within three days and at a four-week followup was pain free. With carditis, the use of corticosteroids is still somewhat controversial but many would give them if congestive

heart failure developed.

3. The third arm of therapy is prophylaxis for recurrent rheumatic fever. The recommended protocol is 1.2 million units of IM benzathine penicillin every four weeks for a minimum of five years after the last attack.¹ If carditis is present, life-long therapy is probably warranted.

Summary

In summary, rheumatic fever seems to be on the rise in the United States. It remains to be seen whether this case is an example of a resurgence of ARF in adults in Virginia, but it probably does, since there are a number of cases being seen in the pediatric population.

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Ankylosing Hyperostosis of the Spine: Case Report

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Washington, DC

THE purpose of this paper is to present a case of ankylosing hyperostosis of the spine (AHS) and review the literature on this rare disease.

Case Report

A 44-year-old retired lieutenant colonel of South African origin was referred to the orthopedic clinic because of limited flexion of neck motion and numbness of each hand. The patient did not have dysphagia. The physical examination showed moderate loss of neck flexion with slight discomfort. The sensory examination was negative. The reflexes of the upper extremity were within normal limits. The patient stated that in the past few weeks the numbness of the hands had subsided, and he was completely asymptomatic.

The glucose tolerance test was negative.

Röntgenogram showed exuberant anterior osteophytes with bony bridges continuous with the anterior vertebral cortex, the anterior longitudinal ligament, and the annulus fibrosus of the intervertebral discs. There was a lack of involvement of the apophyseal joints (Fig. 1). The bony density was within normal limits. The posterior walls of the vertebrae were not

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Fig. 1 (left). Exuberant anterior osteophytes demonstrate bony bridging continuous with vertebral cortex. Figs. 2A (center)



affected. There was no posterior ligamentous ossification. The neural foraminae were clear (Figs. 2A, 2B).

In this case the patient had minimal symptoms and did not require treatment.

Discussion

Forestier and Lagier described AHS in 1971,¹ and it is called "Forestier's disease." It is a distinct entity which may be readily distinguished from osteoarthritis, ankylosing spondylitis, spondylosis, and postinfectious and posttraumatic changes in the vertebrae, although all of these conditions may produce osteophytes.² It is found more frequently in male patients. The onset of symptoms is approximately 50 years.¹

AHS has the following distinguishing features: The bony osteophytic bridges are continuous with the anterior vertebral cortex. The ligamentum longitudinale anterius is partially integrated into the bony proliferations at the level of the vertebral edges and at the pre-disc spaces. The vertebral cortex is thickened but rarely associated with osteoporosis.¹ The disc spaces are normal in height and the vertebral plates are regular in appearance. The apophyseal joints are not involved.¹ The ossifications are not present on the posterior wall of the vertebral bodies as disc spaces.¹ In certain instances, a widening of the spinal processes is noticeable (Bastram syndrome).³ The association of hyperostosis and diabetes mellitus indicated by Boulet has been proven to be frequent.^{4,5} Spinal pain may be absent or moderate. Dysphagia can be present when AHS of the spine involves the cervical spine.

Physical examination reveals diminished spinal motion, particularly in the lower dorsal spine, where the bony bridges are more frequent. Spinal rigidity is never severe and thoracic cage motion is normal. AHS of the spine may involve the cervical, dorsal and lumbar spine.¹

As to differential diagnosis, AHS is to be differenti-

and 2B (right). Neural foraminae demonstrate no osteophytic encroachment on oblique views.

ated from the following four conditions:

1. In spondylosis deformans of Schmorl,⁶ the marginal osteophytes do not lie on the border of the vertebral body. They form on its exterior surfaces at a point where the cartilaginous marginal plate enters in contact with the vertebral body.

2. In osteoarthritis of the spine, the interapophyseal joints are involved.

3. In ankylosing spondylitis,³ the bony bridges are thicker and involve the interapophyseal, costotransverse and sacroiliac joints. The radicular type of pain comes from the lesion of the interapophyseal joints of the cervical spine.

4. In postinfectious and posttraumatic hyperostosis, the hyperostoses are secondary to the adjacent deterioration. In these cases the clinical features are minor, prognosis is good, and no special treatment is necessary.

Summary

Ankylosing hyperostosis of the spine is described as osteophytic spurs or anterior osseous bridges with thickening of the corresponding vertebral cortex. The ossification includes the ligamentum longitudinale anterius and the peripheral part of the disc. Our patient had minor complaints but at the time of consultation was symptomatic.

Gratitude is extended to Miss Melissa Gates and Dr. John Long for their help with this manuscript.

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Bilateral Tubal Pregnancy: Case Report

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Case Report

A 31-year-old black woman, gravida 4, para 0, aborta 3 (two induced and one spontaneous, all first trimester), at 6.5 weeks gestation by dates, presented with vaginal bleeding and lower abdominal cramping of two weeks duration. The patient's beta human chorionic gonadotropin (HCG) level on admission was 1980 mIU/mL, whereas four and seven days earlier, when she was evaluated by the emergency room staff, it was 1950 mIU/mL and 1100 mIU/mL respectively. The patient gave a history of gonorrhea pelvic infection in 1977, as well as repeated Bartholin cysts. She used no contraceptive method and was on no medication. On physical examination the vital signs were stable, abdominal examination revealed no tenderness and no masses, with good bowel sounds. Pelvic examination was positive for mild bleeding per cervical os, normal size uterus, and a right adnexal mass tender to palpation. Left adnexa was normal. Ultrasonography confirmed the pelvic examination, with presence of fluid in the cul-de-sac.

The patient underwent laparoscopy and laparotomy for suspected right ectopic pregnancy and was found to have a right ectopic ampullary gestation of 2 × 1.5 × 1.2 cm in size, and a left ectopic ampullary gestation of 1 × 1.5 × 1.5 cm in size. A right salpingostomy, a left partial salpingectomy, and inspection of intraabdominal structures were done. The patient's postoperative course was uncomplicated.

Pathology revealed partly degenerated large chorionic villi from the right ectopic, and young chorionic villi from the left ectopic. No evidence of inflammatory changes was found on either fallopian tube.

Discussion

Ectopic pregnancy is one in which implantation of the fertilized ovum occurs outside the endometrial cavity. The incidence of ectopic pregnancy in the United States is now about one of every 100 reported pregnancies.¹ Combined pregnancy is a tubal pregnancy that coexists with an intrauterine pregnancy. It

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occurs in about one in 30,000 births¹ and can be complicated by a third pregnancy in the opposite tube.² Twin tubal pregnancy has been also reported, with both embryos in one tube³ or one in each tube. Olive et al reported a bilateral tubal pregnancy following gamete intrafallopian transfer (GIFT).⁴

Theoretically, the incidence of bilateral tubal pregnancy should be the result of multiplying the incidence of twin pregnancy by the incidence of ectopic pregnancy in each tube; that is $1/89 \times 1/100 \times 1/100$, or one pregnancy in 890,000. This value may be clinically smaller, however, since some of the ectopic implantations may die and resolve spontaneously, or more rarely develop into a fetus papyraceous.

Tubal pregnancy remains an uncertain diagnosis, but it should always be part of the differential diagnosis when indicated because of its potentially serious complications. Tubal pregnancy is still responsible for 14% of all maternal deaths.⁵ Tubal causes for ectopic pregnancy, such as chronic salpingitis, remain variable in the literature, ranging from 5% to 90%.^{6,7}

Summary

One important conclusion should be made. When a laparoscopy or laparotomy is done, one should take full advantage of this by inspecting and/or palpating

the second tube, the ovaries, and most pelvic and abdominal structures. Incidental diagnosis may prove to be at times as important as the one for which the surgery is being done.

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ABSTRACTS

These abstracts derive from the scientific session of the annual meeting of the Virginia Dermatological Society on November 12, 1989, in Richmond. Dr. Robert J. Pariser was program chairman.

Yellow Nail Syndrome. Bonnie F. Straka, MD, Charlottesville.

A 74-year-old man was hospitalized because of recurrent pleural effusions, causing shortness of breath, and ankle edema. He had a 10-year history of abnormal fingernails and toenails which showed thickening, yellowish discoloration, slow growth, and increased transverse and longitudinal curvature. There was also some onycholysis and cross-ridging. Hospital workup which included bronchoscopy, thoracentesis, pleural biopsies and CT scans, failed to reveal any evidence of malignancy or infection. In addition, he had weight loss, fatigue, and anorexia. The patient underwent thoracoscopy with talc pleurodesis. Two days later, however, he developed a cold, pulseless left leg which required emergency left femoral endarterectomy, embolectomy and fasciotomy. He deteriorated rapidly thereafter and died several hours later of acute cardiopulmonary arrest.

The yellow nail syndrome in its full-blown form involves a triad of yellow nails, primary lymphedema, and recurrent pleural effusion. These findings are thought to be secondary to congenital hypoplasia of the lymphatics. This full triad is not seen in all patients and early in its development the nail changes may be the only clinical signs. There is no known therapy for the nail disease. Medical therapy centers around control of peripheral edema and management of pulmonary complications.

Wegener's Granulomatosis Presenting with Migratory Polyarthritis and Leucocytoclastic Vasculitis of the Skin. Barbara T. Sitton, MD, Charlottesville.

A 23-year-old man was seen because of a low-grade fever, migratory polyarthritis, and a petechial rash. His past history included a recent tick bite, antibiotic treatment for tooth extraction, and recent episodes of epistaxis. He developed some blistering of the feet which showed no organisms on culture. He was treated with ceftriaxone and doxycycline for a differential diagnosis of gonococcal arthritis vs Lyme disease; however, his arthritic symptoms worsened and the rash became more purpuric. In addition, he developed conjunctivitis with subconjunctival hemorrhage. A skin biopsy at this time revealed leukocytoclastic vasculitis. Prednisone therapy was begun for a diagnosis of serum sickness-like reaction. One week later, he developed sinus tachycardia with primary A-V block. He developed some nasal inflammation and was

found to have evidence of sinusitis on CT scan. Hematuria and proteinuria subsequently developed. Examination revealed a thin young man with tenderness and effusion over the right wrist, right knee and left ankle. There was extensive, palpably purpuric, eruption primarily of the lower extremities and trunk. There was bilateral conjunctivitis with subconjunctival hemorrhage. Biopsy of the skin showed prominent leukocytoclastic vasculitis associated with deposition of IgM and C-3 in the perivascular dermis. Renal biopsy showed segmental sclerosis and necrosis of glomeruli and fibrin deposition. One glomerulus was surrounded by granulomatous inflammation.

Wegener's granulomatosis is characterized by the triad of necrotizing granulomatous lesions primarily of the upper and lower respiratory tract, necrotizing vasculitis of the lungs and sometimes other sites, and renal disease manifested as necrotizing glomerulitis with granulomatous inflammation of the extraglomerular tissue. Continuous lesions are said to occur in up to one half of patients. These lesions consist of necrotic papules and/or nodules, ulcers, and petechial and ecchymotic lesions.

Relapsing Polychondritis. Peter Jaber, MD, Charlottesville.

A 69-year-old man presented with a one-month history of pain and swelling in the left ear and in the nose, unresponsive to treatment with dicloxacillin and prednisone. He had no history of arthralgias, hoarseness, dyspnea, fever or other systemic symptoms. Examination showed marked warmth, redness, tenderness and induration of the left ear with sparing of the ear lobe. There was also mild swelling over the bridge of the nose. Laboratory data included a white count of 15.4 with 82% polymorphonuclear leukocytes, an erythrocyte sedimentation rate of 60, a negative ANA, and negative rheumatoid factor. A biopsy of the left ear revealed chondritis and perichondritis with fibrillar degeneration of the connective tissue. Initial treatment included prednisone 60 mg a day and dapsone gradually increased to 150 mg a day over a 3-week period. When the dose of prednisone was tapered below 40 mg a day, his disease recurred with inflammation and hearing loss in the right ear. A saddle-nose deformity was noted at this time. Dapsone was increased to 200 mg per day but the inflammation persisted. At this point, dapsone was discontinued and cholchicine 0.6 mg bid was added with a continued

prednisone dose of 35 mg a day. On this regimen, he developed definite improvement of symptoms in one week.

Relapsing perichondritis is manifested clinically as recurring inflammation of the cartilage of the ears, nose, and occasionally trachea, sometimes associated with arthritis, ocular inflammation, audiovestibular damage and, less frequently, cardiovascular damage. Approximately 30% of patients have associated connective tissue disease. The disease typically goes through periods of exacerbation and remission. Treatment traditionally centers around the use of prednisone and/or dapsone.

Angiokeratoma Circumscriptum. David M. Pariser, MD, Norfolk.

A white female infant was noted at birth to have large, bulky, vascular tumors on the anterior and medial aspects of the left leg and foot. In some of the areas, particularly the dorsum of the left great toe, there was deep ulceration. Areas of crusting and hyperkeratosis were evident within the lesion. There was no family history of vascular anomalies. Topical therapy with hydrogen peroxide soaks and silver sulfadiazine cream resulted in healing of the ulcerations over a 2-week period; however, the size of the lesion itself remained unchanged.

Four types of angiokeratoma are generally recognized, namely, angiokeratoma circumscriptum, angiokeratoma of Mibelli, angiokeratoma of the scrotum and vulva, and angiokeratoma corporis diffusum (Fabry). The circumscriptum type most commonly presents as a congenital lesion, typically on the lower leg and foot. The lesions have a deep, bluish-red color and sometimes have a linear or zosteriform distribution. Hyperkeratosis and crusting are typically seen. Unlike other hemangiomas of infancy and childhood, these lesions show no particular tendency to involute.

Angiolymphoid Hyperplasia with Eosinophilia. R. B. Henry, III, MD, Norfolk.

A 30-year-old woman developed enlarging nodules behind her left ear. The lesions were painless but did bleed upon minor trauma. In addition, she complained of a pulsating sensation in the ear. Physical exam showed multiple subcutaneous nodules of the left pinna on both front and back surfaces as well as on the adjacent scalp. Clinical diagnosis of angiolymphoid hyperplasia with eosinophilia was confirmed by histologic examination, which revealed dilated thin-walled blood vessels having plump endothelial cells. Stroma contained collections of lymphocytes and many eosinophils. Intralesional steroid injection and piecemeal laser ablation were unsuccessful. En bloc surgical excision is being considered.

Angiolymphoid hyperplasia with eosinophilia manifests as persistent papules and nodules typically of the head and neck region. The ears are a common site.

The lesions have been variously interpreted as inflammatory and as neoplastic. Their behavior is benign. The pathogenesis is obscure. Treatment consists of surgical excision or ablation.

Rhabdomyomatous Mesenchymal Hamartoma. George Elgart, MD, Richmond.

A 3-month-old infant, a healthy boy, was seen for the presence of a "skin tag" that had been present at birth. The lesion was stable and was removed for cosmetic reasons. The nodule, clinically, was soft and mobile without fixation to underlying structures. A histopathologic examination showed a relatively normal epidermis. Pilosebaceous units were present. Centrally, there was a moderate amount of mature adipose tissue. Throughout the dermis, most prominently between the appendages and the adipose tissue, were randomly arranged intersecting bundles and fascicles of striated skeletal muscle. Immunohistochemical examination was positive for the presence of myoglobin, vimentin, desmin, and actin. S-100 protein staining was negative for neural elements.

Rhabdomyomatous mesenchymal hamartoma of the skin was the term given to a recent entity first described in 1989 as a skin tag-like growth of the newborn whose most characteristic histopathologic feature was the presence of skeletal muscle as described above.

Generalized Morphea Versus Progressive Systemic Sclerosis with Bullae. Robert B. Scoggins, MD, James W. Patterson, MD, and Duncan S. Owen, Jr., MD, Richmond.

A 62-year-old woman had a gradually progressive disorder consisting of leg tenderness, particularly in the Achilles region, and swelling of the extremities eventuating in firm skin. She had a 20-year history of bluish discoloration of the fingertips when exposed to cold. Blistering occurred on her right shin and slowly evolved to involve both shins and forearms. She had no signs or symptoms of internal organ involvement. Examination showed tense, firm skin from the dorsum of the feet to the buttocks, with sparing of the medial thighs. The forearms and much of the upper arms were similarly involved. A well-demarcated white plaque was present on the abdomen. There were some vesicles on the right shin. On later examination she showed confluent vesicles and bullae, some of which were hemorrhagic, and linear configuration on both shins and on the dorsum of the forearms. Laboratory studies showed normal hemogram, urinalysis, and routine chemistries. Anti-SCL-70 antibody and rheumatoid factor was negative. ANA was positive at a titer of 1:160 in 1988 although subsequent determinations were negative. A biopsy of the skin of the right leg was taken which showed epidermal acanthosis and hyperkeratosis, sclerosis of the dermis and subcutaneous adipose septa as well as the underlying fascia.

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Focal inflammatory infiltrates in which there were lymphocytes and plasma cells were present in the upper dermis, at the dermal subcutaneous junction, and in the deep subcutis. Likewise, the fascia was diffusely infiltrated with inflammatory cells with some extension into the skeletal muscle. Atrophy of dermal appendages was noted. Eosinophils were not present. Alcian blue stain failed to find any evidence of mucin deposition. Treatment has consisted of prednisone in doses of 7.5–15 mg daily which has produced little clinical response.

Hemorrhagic bullae surmounting lesions of cutaneous morphea have been reported rarely. Such cases raise the issue of the relationship of localized morphea to lichen sclerosis et atrophicus which also can have blistered lesions. In some patients, distinguishing between these two entities may not be possible.

Lymphocutaneous Sporotrichosis. Sharon S. Camden, MD, Richmond.

A 71-year-old woman developed an ulcerated lesion on the dorsum of her right hand. In addition, she had multiple erythematous firm nodules located on the right forearm and upper arm in a lymphatic distribution. The patient gave the history of being a rose gardener and of working with sphagnum moss. No axillary adenopathy was present. A skin biopsy showed the findings of cutaneous sporotrichosis. Organisms were identified on PAS stains.

This most common presentation of cutaneous sporotrichosis occurs as a primary inoculum followed by proximally distributed nodules progressing up a lymphatic chain. Diagnosis is commonly suspected on skin biopsy although organisms are few and may not be identifiable. Diagnosis is confirmed by culture. Classic treatment consists of oral administration of saturated solution of potassium iodide in an increasing dose to tolerance. Treatment usually lasts for about two months or for 30 days after disappearance of lesions.

Pemphigus Vulgaris. Beverly S. Darwin, MD, Richmond.

A 59-year-old black male developed blistering and erosions of the face, scalp, oral mucosa, trunk and proximal extremities. Skin examination showed 1–5 cm weeping, crusted erosions with peripheral borders of tense, flaccid bullae. Nikolsky's sign was negative. The oral mucosa had superficial bullae and erosions. Skin biopsy showed suprabasal acantholysis of the epidermis and direct immunofluorescence revealed the presence of IgM, IgG, IgA and C-3 in the intercellular spaces between keratinocytes. Indirect immunofluorescence showed the presence of circulating antibody to the intercellular region of epithelium in a titer of 1:2560. Initial treatment consisted of prednisone 90 mg per day with gradual resolution of the lesions. This dose was reduced to 60 mg per day, at which point

azathioprine 50 mg per day was added as a steroid-sparing measure. Two months after onset of therapy, his circulating antibody titer had dropped to 1:320. After five months of therapy, his prednisone dose was 20 mg a day and the azathioprine dose continued at 50 mg per day.

Pemphigus vulgaris is a classical autoimmune primary bullous disease of the skin which confers a high mortality if untreated. Systemic corticosteroids are life-saving. More recently, treatment has tended to consist of immunosuppression plus steroids, which seems to allow for reduction of steroid dose and side effects. Many patients, if maintained relatively lesion-free, will enter periods of remission after a number of years.

Pityriasis Rubra Pilaris. Neeraja C. Matty, MD, Richmond.

A 56-year-old white man developed redness and fine scaling of the scalp and face 2 weeks after an episode of contact dermatitis. For the next month, he noted gradual thickening and scaling of the palmar and plantar skin. Soon thereafter, he developed a generalized erythroderma. Examination showed salmon-colored erythema with islands of normal appearing skin. There was scaling over the scalp, face and extremities. There was yellowish palmoplantar hyperkeratosis. The nails showed subungual hyperkeratosis. The mucosa was normal and there was no lymphadenopathy. A skin biopsy showed horizontal and vertical alternating orthokeratosis and parakeratosis of the upper dermis. Treatment initially consisted of oral methotrexate 5 mg a day for 5 days followed by 2 days off drug. Initial improvement was noted, but after completing three such courses, the patient developed fever of unknown origin with signs of high output heart failure. He died shortly thereafter.

Pityriasis rubra pilaris is a relatively rare papulo-squamous disease of unknown cause which, in its full-blown adult form, shows a widespread symmetric salmon-colored erythroderma associated with hyperkeratosis of the palms and soles and sometimes follicular lesions. Although typically grouped in the category of psoriasis-like conditions, it is much more recalcitrant to therapy than is psoriasis. The course tends to be prolonged.

Squamous Cell Carcinoma of the Lip. Ricardo G. Mora, MD, Norfolk.

A 25-year-old white man developed a rapidly growing lesion of the lower lip. He was a cigarette smoker. Examination showed a 1.5-cm granulomatous indurated lesion of the left lower lip. There was no palpable lymphadenopathy. A skin biopsy revealed squamous cell carcinoma. The patient underwent Mohs micrographic surgery in three stages involving seven sections, producing a resultant defect of 3.0×1.5 cm. Following plastic surgery consultation, it was decided

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to allow the wound to heal by secondary intention. Satisfactory healing was achieved in 4 weeks.

Squamous cell carcinoma of the lip is a potentially lethal tumor, carrying a much more grave prognosis than the more common squamous cell carcinoma of sun exposed skin. Standard surgical excision using wedge resection gives an 87% 5-year cure rate. Radiation therapy has been reported to result in cure rates of 77%. The cure rate of a large series of cases done by Mohs micrographic surgery is 94%. Lymphatic involvement is said to be present in 18% of lesions greater than 2 cm. This patient's young age was unusual for this lesion, emphasizing the fact that any new or persistent lesion of this area should be diagnosed by biopsy.

Two Cases of Atrophoderma of Pasini and Pierini. Robert J. Pariser, MD, Norfolk.

A 36-year-old black woman was seen because of the development of an asymptomatic lesion of the right posterior shoulder three weeks previously. Her past medical history was unremarkable. She denied any injections or trauma to the affected area. Examination showed a 2-cm sharply defined area of atrophy and hypopigmentation which was round in shape and showed a well-defined "cliff-drop" border. The lesion was soft and pliable. A skin biopsy showed reduction in normal dermal thickness with normal collagen and no inflammatory infiltrate. No treatment was given. Followup by telephone seven years and nine months later indicated the lesion had gradually healed, leaving a flat scar.

A 32-year-old black woman noted a gradual onset of lesions on her back. The lesions were asymptomatic. There had been gradual extension from the original site on the right upper back to the right shoulder and the right arm. Her past medical history was unremarkable. There was no history of trauma or injection to the area. Examination showed multiple ovoid and linear areas of sharply defined indentation of the skin with a "cliff-drop" edge. Many of the lesions were hyperpigmented. The texture of the skin was normal. There was no evidence of sclerosis. A previous medical workup had shown no systemic abnormalities. Biopsy of the affected area and of the adjacent normal skin showed thinning of the affected dermis. There was a mild superficial perivascular inflammatory infiltrate. The size, staining and patterning of collagen bundles was normal.

Originally described by Pasini in 1923 and Pierini in 1936, this entity usually presents as asymptomatic single or multiple areas of dermal atrophy. Unlike localized scleroderma, the affected skin is soft and pliable. No evidence of sclerosis, except perhaps late in the course of the disease when some lesions appear to heal with scarring. An unusual variant of the condition involves linear lesions as seen in the second patient noted above. There is no apparent relationship

to systemic disorders. No treatment has been reported successful in arresting the process.

Post-Traumatic (Surgical) Blistering of Unknown Etiology. Ricardo J. Mora, MD, Norfolk.

A 68-year-old white woman underwent Mohs micrographic surgery because of a Bowenoid basal cell carcinoma of the left leg which had been present for two years. The resulting defect was 3.75×2.75 cm following three stages of excision and nine sections. The defect was closed with a split thickness skin graft from the left anterior thigh. Six weeks postoperatively she developed blistering at the superior edge of the previously well-healed donor site. This was resolved spontaneously but recurred approximately two weeks later. Two days later she developed vesicopustulation at the inferior edge of the graft which resolved in one week with compresses. Four months later additional recurrences were noted. Subsequently, she had done well with no further recurrences. Due to previously used Polysporin® ointment, she was patch tested to look for possible contact allergy but none was found. Cultures and stains from the lesions have been negative. A biopsy revealed sub/intraepidermal blistering with eosinophilia. Direct immunofluorescence was negative for IgA, IgM, IgG and complement.

Localized blistering at a surgical site could be due to contact dermatitis, localized bullous pemphigoid, lymphatic obstruction or other localized vesicobullous disease. No specific etiology could be implicated in this case.

Generalized Plane Xanthomatosis With Monoclonal Gammopathy. Kenneth E. Greer, MD, Charlottesville.

An 82-year-old white woman developed gradually progressive yellowish-flat lesions of the face and neck over a 3-year period. The lesions slowly progressed to involve an extensive area of the chest and upper back. They were asymptomatic. The patient had a normal serum lipid profile but was found to have an IgG Kappa monoclonal gammopathy. She showed no clinical response to lovastatin 20 mg daily and prednisone 20 mg daily. A skin biopsy showed scattered mononucleated and multinucleated xanthoma cells in the superficial dermis. Her cholesterol level was 176 and triglycerides 70. Immunofluorescence revealed complement and immunoglobulin within some of the lipid deposits. Bone marrow examination was normal.

Disseminated xanthomatosis of several types, including plane xanthomas, has been associated with monoclonal gammopathy. In some cases, this appears to be a benign serologic finding; in others, the association has been with myeloma. Treatment in such patients has been disappointing.

VIRGINIA MEDICAL

Changes

THIS started out to be a short editorial on the changing faces of medicine in Virginia, with mention of a few of those recently deceased or who have retired from practice. Achievement of this goal is just not possible. Mention might be made of a few of those retiring in Richmond—Wash Winn, Emmett Mathews, Joe Parker, Walter Bundy, Herb Park, Dennis Morey, and Joe Coxe, as well as a sprinkling of others throughout the state as mentioned elsewhere in this issue. Among those recently deceased are John Lynch, Jim Blades, Colon Rivadeneira, and Peter Pastore, all of Richmond; Epes Harris, Blackstone; Bob Neu, Arlington; and Cully Lippard, Lynchburg. And the above constitute only a few of those no longer in practice in Virginia.

For many of those retiring from practice the wrench

has not been too great. The practice of medicine today has become a business, pure and simple. The physician is now a "health care provider." Patients are not always friends; often they present as adversaries. Hospitals, in former days the physician's workshop, are a different entity; Blue Cross-Blue Shield, "the doctor's insurance plan," can no longer qualify as such. Medicare and Medicaid present progressively stricter rules, with ever lower renumeration to the practicing physician.

There is a light at the end of the tunnel. Medicine in the United States still is the best in the world. If you were among those who attended the symposium on "Health Care in Other Countries," recently sponsored by St. Mary's Hospital, you are encouraged. Keep working!

E.L.K., JR.

The Hawk

IT was a hot day in early summer. I had been working so long and so hard in the weedy garden that I was exhausted and soaked with sweat. To cool off I lay on the grass in the shade of the Montmorency cherry tree. As I gazed into the hot, cloudless, blue sky, I saw the hawk gliding and soaring in the air currents high above me.

I was fascinated by its uncanny ability to find updrafts so that with effortless grace it could soar over the countryside. And as I watched, I remembered that birds of prey have extraordinarily keen sight plus two foveas in each eye, twice as many as humans.

Not only do birds of prey have exceptional vision to spot their quarry, but they also have a panoramic view

of the world. They have binocular vision directly in front of them with only a small blind area directly behind them.

As I cooled and contemplated, I wondered if their eyesight might be so good that they literally could see into us. I wondered if they could see through our veneer of civility and into our core, our id, that secret place of secret places where our crassness, rudeness, selfishness, thoughtlessness, dark thoughts and hostility reside.

If I knew the answer, I would know how opaque my gardening clothes and cap should be.

H. S. CAMPELL, MD

How We Got Here: Case Report From the Class of 1904

EARLY in this century G. B. Shaw said, "We have not lost faith, but we have transferred it from God to the medical profession." Paul Starr recently gave a memorable subtitle to his book, "The rise of a sovereign profession and the making of a vast industry."¹ Starr's book charted the large forces behind the ascendancy of the profession between 1900 and 1980: scientific progress in control of many diseases, urbanization and rising public expectations, and distribution of medical costs by insurance. Another of these forces was reputed to be a decrease in the number of medical schools and students early in the century, achieved by raising academic standards, either because of scientific ideals or monopolistic design, depending on different readings of the same data.

Because history includes both the tracing of such large forces, and individual and personal views from the time, I offer this recorded interview about medical education at the University of Virginia with my uncle, Dr. H. Norton Mason of Richmond, who was 94 years old at the time of our conversation.

Uncle Norton entered the University of Virginia in 1900 and received the MD degree in 1904. At that time there were relatively few formal requirements in terms of premedical courses, no MCAT and less competition, and the doctorate could be reached by two years of classes in medical sciences after some evidence of basic preparation.

Uncle Norton had probably prepared for our interview, as suggested by his careful recitation of the eight faculty members and their topics of instruction.² The number of MD's awarded annually at the University of Virginia at that time was about 20, an interesting ratio of students to faculty. In 1985 there were 566 members of the full-time faculty in both basic and clinical areas, and each class in the 4-year curriculum had about 140 students.

There were no federal grants and small state support. The first hospital at the University of Virginia opened in 1902. Dr. Mason recalled lectures and laboratory exercises with microscope in the Anatomical Theater, designed by Thomas Jefferson and demolished in 1938 to make way for the Alderman Library. At this time in this medical school the instruction in the preclinical sciences appears to have excelled that

in the practical areas: "We were very well trained didactically. Our students were so successful in New York City at Bellevue that almost all of them were given appointments, and a complaint went up. Finally, Bellevue made a rule that the University would be allowed only a certain portion of appointments." After the establishment of a State Board of Medical Examiners for licensing physicians in 1885, "the University had a record of practically no failures."

Dr. Mason's memory of practical instruction suggests lean and informal fare, focused on the Dispensary rather than the new hospital: "Well, when I first went there the hospital consisted of an old brick building directly across the street from the Dispensary Building." (These buildings were located on both sides of Main Street in approximately the areas of the current University Theater and the George Rogers Clark Monument.) "The Dispensary building had an amphitheater. The patients waited out in the hall and kept warm in the winter by a large pot-bellied stove. Each clinical professor had appointed one of the students as his assistant, who lived in the dispensary and got his room free that way.

"I might mention a picturesque old janitor named Jack Collier, who slept in the building. The Dispensary did not have a sterilizer. When Jack felt like things ought to be a little cleaner he would take a bucket of hot water and pour it over the spigot, and he said, 'There doctor, that kills them all', apparently meaning the source of infections.

"The patients did not stay in the Dispensary overnight. The patients in the hospital across the street were anesthetized and brought over on a stretcher into the amphitheater, where there was an old operating table you might call it, built by a local carpenter. It rotated so that the students could see the operations. The surgeons brought their own instruments with them, already boiled at home and wrapped in a sterile towel. There was little in the way of practicing asepsis. After these operations patients were carried back to the so-called hospital".

"The members of the University faculty and other city doctors did the major operations in the patients' homes in those days, using the kitchen table as an operating table, and apparently they had very good

The operating room in the Dispensary, with rows of seats for student observers. Note the absence of surgical masks.



results." Although I failed to ask Uncle Norton to specify, it appeared that the dispensary was used more for rural and less prosperous patients.

"No obstetrical patients ever went to hospitals in those days. It was all done at home, and the students at the session on obstetrical teaching were very anxious to have experience. We paid a special black woman one dollar for getting us each a case of obstetrics. The material consisted of going to the drugstore and getting ten cents worth of creolin, and some bought a five-cent nail brush, and the results were very good."

In the 1980s medical education has seen debates about the effectiveness of the clinical faculty, whose primary interest may often be biomedical research, and the need for "role models" has been raised. I asked Uncle Norton whether he thought of his faculty as having been good teaching physicians or surgeons.

"Well, I don't like to go into particulars," he answered, "but in those days a doctor was not considered a very fine doctor unless he did his own surgery. The doctors who did not operate were looked down on by the general public as not 'finished' doctors, who didn't know how to operate on patients. At the new University Hospital, which was built later on, they did not allow surgeons in the city to operate unless they were teaching at the hospital."

"Some of the city doctors were good teachers, especially Dr. Magruder. He was a very good teacher on physical examinations, and in fact he wrote a book about it. Dr. Browning was a very good surgeon. During my third year in medicine Dr. Charles Venable

of Charlottesville, whose father was a professor at the University, gave me a position as his assistant. I had an opportunity to go around with the men around the city. I lived in Dr. Venable's home and used his father's old office for a bedroom. Dr. Venable decided that he and Dr. Browning would go North for two or three weeks to study. He said, 'Now you are entirely in charge of my practice while I am gone.' I had not even studied the practice of medicine then, but I told him I would do the best that I could. The few patients that came in, I would talk to them and examine them a little, and then get out Squibb's *Materia Medica*. It is full of prescriptions, and I would find the page with the name of the disease and copy a prescription. I don't know whether I did any good, but I don't think I killed anybody."

THE HIGH IMAGE of surgery reflected in Dr. Mason's comment was, as we now know, shared increasingly by society in our time. Surgeons had been on a long climb from a primary affiliation with barbers in the 17th century, and the 20th century has seen remuneration patterns and even excessive procedures as targets for reform. The curriculum committees of today are realizing that entirely hospital-based teaching gives students a distorted emphasis on severe illness and intensive use of technologies. They plan to put students more in "ambulatory care settings" and perhaps again use the resources of volunteer faculty. Dr. Mason's experience may give hope to those attempting this difficult change. Delegation of responsibility, such as Dr. Venable could give

his student assistant, and considered invaluable for student learning, is riskier when medicine's power both to help and to harm are much greater.

In 1904 there was almost no support for postgraduate training, which was recognized then as especially important for graduates of a school in a rural area without a substantial hospital. Accordingly, it was important to gain practical experience by spending a year or two in an urban area where population density and disease frequency gave better chances at observation and responsibility. Dr. Mason noted that after medical school he "had no money", and he first took a position at Central State Hospital in Petersburg, primarily a custodial institution for the mentally ill.

"Then I went to St. Luke's Hospital in New York for the summer until I got a position on Randall's Island at the hospital for children, which was owned by the city. By the way, on this island were 2,500 employees, and most of them had been run out of the city courts, the judge saying, 'You can go to jail or you can to go Randall's Island and work for ten dollars a month.' There were all sorts of hospitals and dispensaries in New York. I was always eager to get some practical training in studying patients. Dr. Berkely, a native of Staunton, ran a course of one month at the Good Samaritan Hospital down in the slums. Nobody spoke English, but he was a very good teacher, and I got a great deal of information from him.

"Most hospitals had their own ambulances. They were all horse-drawn, and they were open in front and back. No front windows or back doors, and in zero weather the zero weather blew right through. At the New York Infant Asylum, where I spent most of my residency, the health department rule was that any child who developed a contagious disease was sent up the river to another city hospital. Many of them died up at what was called the Riverside Hospital, which was a pretty bad situation.

"There was a great deal of diphtheria of the larynx, called membranous croup. There was a hospital called the Willard Parker Public Hospital, and they had two ambulances, and they carried the instrument called intubation. The hospital would get these hurry "croup calls," and they would be there in a very short time. The intern and the driver would rush up to the room and put the baby on the kitchen table, and intubation would be done in a few seconds."

UNCLE NORTON returned to Richmond to start an independent practice of general medicine and pediatrics in 1907. He started a pediatric clinic at the Medical College of Virginia for indigent patients. His office was the front room of his home, a handsome brick house (since demolished) at the corner of Lombardy and Grace Streets. The waiting room was the house's front hall, and I remember passing through the waiting room in the 1940s to visit the family, who congregated to the rear and upper floors of the house.

In the front hall waiting room would be a row of patients—someone with an abscess to be drained, another with a cough or rash to be treated, an infant in arms with who-knows-what, and a worker with a recent fracture to be set and casted. The nature of these problems were then unknown to me, but the frosted glass pane on Dr. Mason's office-surgery door and the smell of ether made me think something important was happening on the other side. There were no hints of great prosperity in the family—no expensive cars, clothes, hobbies or vacation trips. These amenities may now be taken for granted by the doctors of our time. Then, the opportunity to practice the craft must have meant both burden and satisfaction enough.

The most challenging time was the influenza epidemic of 1918.

"This time they opened up the windows, giving them the fresh air treatment, and an enormous amount of pneumonia occurred from breathing this cold air. The John Marshall High School here was turned into a hospital, and they were brought in alive through the front door and carried out dead through the back door. I went down there and told them to pull down all the windows . . . When I was at the University, Dr. John Staige Davis one day in his lecture stated that there was a specific for influenza, and this was a drug called bicarbonate of potassium, and the dose was 30 grams dissolved in water every two hours . . . During that and many other years afterward I lost practically no patients."

I asked Dr. Mason for a summary of the changes in the medical profession, or the public's perception of us, during his lifetime.

"Doctors were often a friend of the family. As I have mentioned, people would tell their doctors things that they would not tell their minister . . . There were very few hospitals when I started out in 1907. The Memorial Hospital at 12th and Broad was a model, up to date, partly private and partly wards. The rate in the ward was one dollar per day, which included bed, general nursing, meals and medicine. No charge for the operating room or laboratory. Very few people had any money to go to the hospital, they didn't have the dollar a day to pay. The single rooms were twenty-five dollars a week, and a bed in a double room was fifteen dollars a week. The nurses in those days were mostly very fine country girls, many of them daughters of physicians. They had a purpose to dedicate their lives to the profession, and hours were not considered. I think that there was so much dedication in the first part of my life as a doctor, selflessness among nurses and doctors. I don't think that more than 50% of the patients that I had were paying patients. The doctors treated all those that didn't have money free of charge."

"Did people try to take advantage of you?"

"No, not at all."

THESE RECOLLECTIONS probably contain minor errors in facts, and our main task is to decide the reality of the interpretive comments. American medicine has been described recently as coming more from an entrepreneurial than a service tradition; the commentators and participants of two generations ago seem to have recognized some elements of a service tradition now less visible. We live in a time of greater cultural diversity, greater confidence in technology and the therapeutic paradigm, less durable affinities among persons and less discrete roles of institutions, greater material rewards, and possibly fewer psychological rewards among the medical profession.

Much of the 20th century thought in the West has been dominated by a therapeutic or educational paradigm—the solving of problems by technological or instructional interventions. Disenchantment with this recently may be occurring, and may be in part a realistic perception of our limits. Nevertheless, the therapeutic paradigm based on biomedical science and properly applied, deserves to endure. Its exploitation either for personal gain or in ignorance of its limits will weaken it. If the students of recent years have shown an entrepreneurial focus or preoccupation with the therapeutic paradigm, perhaps substitution in the curriculum of history and literature would broaden their vision. Along with the current emphasis on "problem-solving skill," there should perhaps be some cultivation of concept development and felicity of communication.

We have heard both ominous portents and hopeful initiatives from medical educators. Dr. Mason's case report, roughly congruent with many other observations from and about his time, may give clues to some of the directions, where an advance may be, without apology, the same as a recovery.

LOCKHART B. MCGUIRE, MD

¹ Paul Starr, *The Social Transformation of American Medicine*, 1982

² Listed by Dr. Mason were Dr. Buckmaster, professor of Obstetrics/Gynecology; Dr. Paul Barringer, professor of physiology and *materia medica*; Dr. John Staige Davis, professor of the practice of medicine; professor of chemistry John W. Mallet; Dr. William G. Christian, professor of anatomy and surgery; Dr. Albert Tuttle, professor of biology; and assistants Dr. James B. Bullitt and Dr. Carroll Flippin. The University of Virginia catalog for 1904 lists in addition to these full-time faculty six part-time clinical faculty members, several of whom are described in this interview. There were 30 doctorates in medicine awarded in 1904.

A complete transcript of Dr. McGuire's interview with Dr. Mason appeared in the Winter 1976 edition of the *University of Virginia Medical Alumnews*.

Address correspondence to Dr. McGuire at Box 158, University of Virginia Health Sciences Center, Charlottesville VA 22908.

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OBITUARY

- **Robert B. Neu, MD**, Arlington psychiatrist; Georgetown School of Medicine, 1942; age 69; died March 16, 1990. A past president of the Arlington Medical Society, he had been Speaker of the Assembly of the American Psychiatric Association.
- **Raymond Schwartz, MD**, longtime Falls Church internist; Cornell University School of Medicine, 1937; age 76; died March 24, 1990. He had been chief of the health services division of the Arlington Department of Human Resources.

Memoir of Frank Sloop 1947-1989

By Stephen E. Schmitz, MD

Frank B. Sloop, Jr., MD, died on August 10, 1989, at the age of 42.

Born in Salisbury, North Carolina, he studied at the University of North Carolina at Chapel Hill where he received a BA in English and a doctor of medicine degree in 1973. He continued his postgraduate medical training at the University of Virginia Medical Center, where he was a resident in pathology 1973-1977.

Dr. Sloop became the first director of anatomic and clinical pathology at the Martha Jefferson Hospital in Charlottesville, Virginia, in 1977. He served in this capacity until his untimely death. His dedication to developing and expanding laboratory services at the Martha Jefferson Hospital was never-ending. His energy and resourcefulness were quickly recognized and he served the hospital in many capacities, including numerous committees and, ultimately, membership in the Health Service Corporation board of trustees and Foundation board of trustees.

Frank was a member of all the major pathology societies, the AMA, and the Albemarle County Medical Society. Through the years he was a recipient of many awards, including the Morehead Award at the University of North Carolina in 1965; Phi Beta Kappa in 1969, several undergraduate honor societies, and Alpha Omega Alpha.

In addition to his mother, Mrs. Gladys Eudy Sloop, Dr. Sloop is survived by his wife, Anne Dye Sloop; a daughter, Katherine Anne Sloop; two sons, Mark David Sloop and Peter Daniel Sloop, all of Charlottesville; a sister, Linda Sloop Nunalee of Wilmington, North Carolina; and a brother, David Arthur Sloop of Raleigh, North Carolina.

Frank is deeply missed by his family, medical colleagues and many friends. His expertise extended far beyond medicine and he was equally respected with a golf club in hand. He accomplished much in a short time and managed to keep all those around him smiling with his quick wit. All who knew Frank B. Sloop, Jr., have suffered a great loss.

Memoir of E. N. Weaver 1917-1989

By Charles B. Bray, Jr., and
Robert S. Hutcheson, MD

Edgar Newman Weaver died in Roanoke, Virginia on August 14, 1989, at the age of 71. He was a true Virginian, born in Orange County on November 13, 1917, the son of Dr. and Mrs. Delmar Franklin Weaver. He attended Hampden-Sydney College and graduated from the University of Virginia School of Medicine in 1943. He served two years in the Navy as chief of neurological surgery at the Bethesda Naval Hospital. After his naval service he returned to his beloved University as clinical professor of neurosurgery, where he remained until starting private practice in Roanoke in 1950. He remained closely associated with the Department of Neurological Surgery while maintaining an active private practice.

His contributions to the field of Neurosurgery were many. He served as chief of neurological surgery and of the neurosurgery intensive care unit at the Roanoke Memorial Hospital for a number of years. He was a member of the board of trustees of Jefferson Hospital and active as consultant at the Veterans Administration Hospital in Salem and the Community Hospital of Roanoke Valley. He was co-founder of the Congress of Neurological Surgery and a member of the Harvey Cushing Society. He was a founding member and former president of the Neurological Society of Virginia. He was a member of the Roanoke Academy of Medicine, The Medical Society of Virginia, and the AMA. He was a fellow of the American College of Surgeons and a diplomate of the American Board of Neurological Surgery and was also a frequent contributor to the Neurosurgery literature. He was a charter member of the Advisory Committee of the University of Virginia School of Medicine, later serving as president. He was a member of the board and president of the University of Virginia Medical Foundation and a member of the White-Burkett Miller Center. For the past six years he was a devoted member of the University of Virginia Board of Visitors and served untiringly from 1983 until his death. He was a member of St. John's Episcopal Church and had served as a vestryman.

He is survived by his wife, Evelyn Richards

Weaver, Roanoke; one son, Dr. Edgar Newman Weaver, Jr., Roanoke; two daughters, Mrs. Margaret Weaver Crosson, Roanoke, and Mrs. Dabney Weaver Dwyer, Dallas, Texas; one brother, two sisters and eight grandchildren. He was preceded in death by his son, Dr. David Delmar Weaver, a neurosurgeon in Dallas, Texas.

Dr. Weaver was a master diagnostician, a gifted surgeon and a caring physician. His family, his host of friends and his patients knew his extraordinary sensitivity and the gentleness of his nature. So many dimensions of his life set high standards for the rest of us and each one of us will treasure our memories of Tuffy and the ways he touched our lives.

versity of North Carolina. He was an honor graduate of the University of Maryland School of Medicine in 1945, being ranked number two in his class. After serving in the Army Medical Corps for three and one-half years, he joined the Melrose Clinic in Roanoke in 1948 and practiced family medicine there for 40 years. This devoted physician whose life was truly one of service, said, while ill, "I have no regrets, for I did my best and if given the opportunity, I would do the same again." He felt so fortunate and proud to be on the staff of such an outstanding hospital as Roanoke Memorial for all those 40 years.

Bill married his wife Jay in 1946, and their children, William L. Foster, Jr., MD, of Raleigh, North Carolina, and L. Annette Foster, of Durham, North Carolina, brought him much happiness. He was proud of his family and their accomplishments.

Bill will long be remembered by his many friends as an avid reader, especially interested in business and history. His real relaxation was boating at Claytor Lake, and some of his happiest times were spent with his grandsons, Mark, Scott and David.

For all his years of dedicated service, he will be remembered fondly by his patients, colleagues, and friends in the community. He was a good friend, devoted to the practice of medicine and to his family.

Memoir of William Foster 1922-1989

By Andrew A. Slemp, MD

William Leicester Foster, MD, of Roanoke, a practicing physician of family medicine, died on June 27, 1989, at the age of 67.

Dr. Foster was born in Asheville, North Carolina, and attended Johns Hopkins University and the Uni-

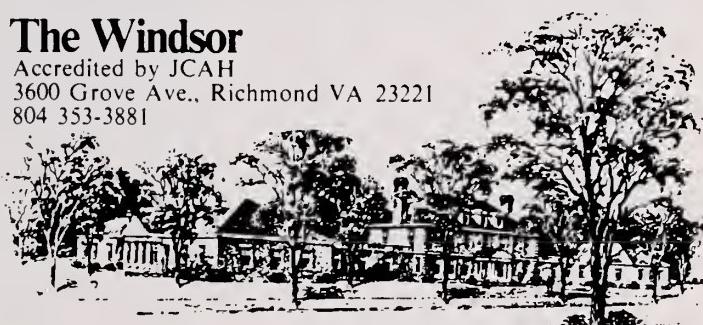


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continued on page 224

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References

1. USP DI Update, September/October 1988, p 120.
2. Br J Clin Pharmacol 1985; 20: 710-713.
3. Data on file, Lilly Research Laboratories.
4. Scand J Gastroenterol 1987; 22(suppl 136): 61-70.
5. Am J Gastroenterol 1989; 84: 769-774.



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Precautions: General— 1. Symptomatic response to nizatidine therapy does not preclude the presence of gastric malignancy.

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Drug Interactions—No interactions have been observed with theophylline, chlordiazepoxide, lorazepam, lidocaine, phenytoin, and warfarin. Axid does not inhibit the cytochrome P-450 enzyme system; therefore, drug interactions mediated by inhibition of hepatic metabolism are not expected to occur in patients given very high doses (3,900 mg) of aspirin daily. Increased serum salicylate levels were seen when nizatidine, 150 mg b.i.d., was administered concurrently.

Carcinogenesis, Mutagenesis, Impairment of Fertility—A two-year oral carcinogenicity study in rats with doses as high as 500 mg/kg/day (about 80 times the recommended daily therapeutic dose) showed no evidence of a carcinogenic effect. There was a dose-related increase in the density of enterochromaffin-like (ECL) cells in the gastric oxytic mucosa. In a two-year study in mice, there was no evidence of a carcinogenic effect in male mice, although hyperplastic nodules of the liver were increased in the high-dose males as compared with placebo. Female mice given the high dose of Axid (2,000 mg/kg/day, about 330 times the human dose) showed marginally statistically significant increases in hepatic carcinoma and hepatic nodular hyperplasia with no numerical increase seen in any of the other dose groups. The rate of hepatic carcinoma in the high-dose animals was within the historical control limits seen for the strain of mice used. The female mice were given a dose larger than the maximum tolerated dose, as indicated by excessive (30%) weight decrease as compared with concurrent controls and evidence of mild liver injury (transaminase elevations). The occurrence of a marginal finding at high dose only in animals given

an excessive and somewhat hepatotoxic dose, with no evidence of a carcinogenic effect in rats, male mice, and female mice (given up to 360 mg/kg/day, about 60 times the human dose), and a negative mutagenicity battery are not considered evidence of a carcinogenic potential for Axid.

Axid was not mutagenic in a battery of tests performed to evaluate its potential genetic toxicity, including bacterial mutation tests, unscheduled DNA synthesis, sister chromatid exchange, mouse lymphoma assay, chromosome aberration tests, and a micronucleus test.

In a two-generation, perinatal and postnatal fertility study in rats, doses of nizatidine up to 650 mg/kg/day produced no adverse effects on the reproductive performance of parental animals or their progeny.

Pregnancy—Teratogenic Effects—Pregnancy Category C—Oral reproduction studies in rats at doses up to 300 times the human dose and in Dutch Belted rabbits at doses up to 55 times the human dose revealed no evidence of impaired fertility or teratogenic effect; but, at a dose equivalent to 300 times the human dose, treated rabbits had abortions, decreased number of live fetuses, and depressed fetal weights. On intravenous administration to pregnant New Zealand White rabbits, nizatidine at 20 mg/kg produced cardiac enlargement, coarctation of the aortic arch, and cutaneous edema in one fetus, and at 50 mg/kg, it produced ventricular anomaly, distended abdomen, spina bifida, hydrocephaly, and enlarged heart in one fetus. There are, however, no adequate and well-controlled studies in pregnant women. It is also not known whether nizatidine can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Nizatidine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers—Studies in lactating women have shown that 0.1% of an oral dose is secreted in human milk in proportion to plasma concentrations. Because of growth depression in pups reared by treated lactating rats, a decision should be made whether to discontinue nursing or the drug, taking into account the importance of the drug to the mother.

Pediatric Use—Safety and effectiveness in children have not been established.

Use in Elderly Patients—Healing rates in elderly patients were similar to those in younger age groups as were the rates of adverse events and laboratory test abnormalities. Age alone may not be an important factor in the disposition of nizatidine. Elderly patients may have reduced renal function.

Adverse Reactions: Clinical trials of varying durations included almost 5,000 patients. Among the more common adverse events in domestic placebo-controlled trials of over 1,900 nizatidine patients and over 1,300 on placebo, sweating (1% vs 0.2%), urticaria (0.5% vs <0.01%), and somnolence (2.4% vs 1.3%) were significantly more common with nizatidine. It was not possible to determine whether a variety of less common events was due to the drug.

Hepatic—Hepatocellular injury (elevated liver enzyme tests or alkaline phosphatase) possibly or probably related to nizatidine occurred in some patients. In some cases, there was marked elevation (>500 IU/L) in SGOT or SGPT and, in a single instance, SGPT was >2,000 IU/L. The incidence of elevated liver enzymes overall and elevations of up to three times the upper limit of normal, however, did not significantly differ from that in placebo patients. Hepatitis and jaundice have been reported. All abnormalities were reversible after discontinuation of Axid.

Cardiovascular—In clinical pharmacology studies, short episodes of asymptomatic ventricular tachycardia occurred in two individuals administered Axid and in three untreated subjects.

CNS—Rare cases of reversible mental confusion have been reported.

Endocrine—Clinical pharmacology studies and controlled clinical trials showed no evidence of antiandrogenic activity due to nizatidine. Impotence and decreased libido were reported with equal frequency by patients on nizatidine and those on placebo. Gynecomastia has been reported rarely.

Hematologic—Fatal thrombocytopenia was reported in a patient treated with nizatidine and another H₂-receptor antagonist. This patient had previously experienced thrombocytopenia while taking other drugs. Rare cases of thrombocytopenic purpura have been reported.

Integumentary—Sweating and urticaria were reported significantly more frequently in nizatidine- than in placebo-treated patients. Rash and exfoliative dermatitis were also reported.

Hypersensitivity—As with other H₂-receptor antagonists, rare cases of anaphylaxis following nizatidine administration have been reported. Because cross-sensitivity among this class has been observed, H₂-receptor antagonists should not be administered to those with a history of hypersensitivity to these agents. Rare episodes of hypersensitivity reactions (eg, bronchospasm, laryngeal edema, rash, and eosinophilia) have been reported.

Other—Hyperuricemia unassociated with gout or nephrolithiasis was reported. Eosinophilia, fever, and nausea related to nizatidine have been reported.

Overdosage: Overdoses of Axid have been reported rarely. If overdosage occurs, activated charcoal, emesis, or lavage should be considered along with clinical monitoring and supportive therapy. Renal dialysis for four to six hours increased plasma clearance by approximately 84%.

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Description: Yohimbine is a 3a-15a-20B-17a-hydroxy Yohimbine-16a-carboxylic acid methyl ester. The alkaloid is found in Rubaceae and related trees. Also in Rauwolfia Serpentina (L) Benth. Yohimbine is an indolalkylamine alkaloid with chemical similarity to reserpine. It is a crystalline powder, odorless. Each compressed tablet contains (1/12 gr.) 5.4 mg of Yohimbine Hydrochloride.

Action: Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalamic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it, however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

Indications: Yocon® is indicated as a sympatholytic and mydriatic. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral α-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.^{1,2} Also dizziness, headache, skin flushing reported when used orally.^{1,3}

Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.^{1,3,4} 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to 1/2 tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.³

How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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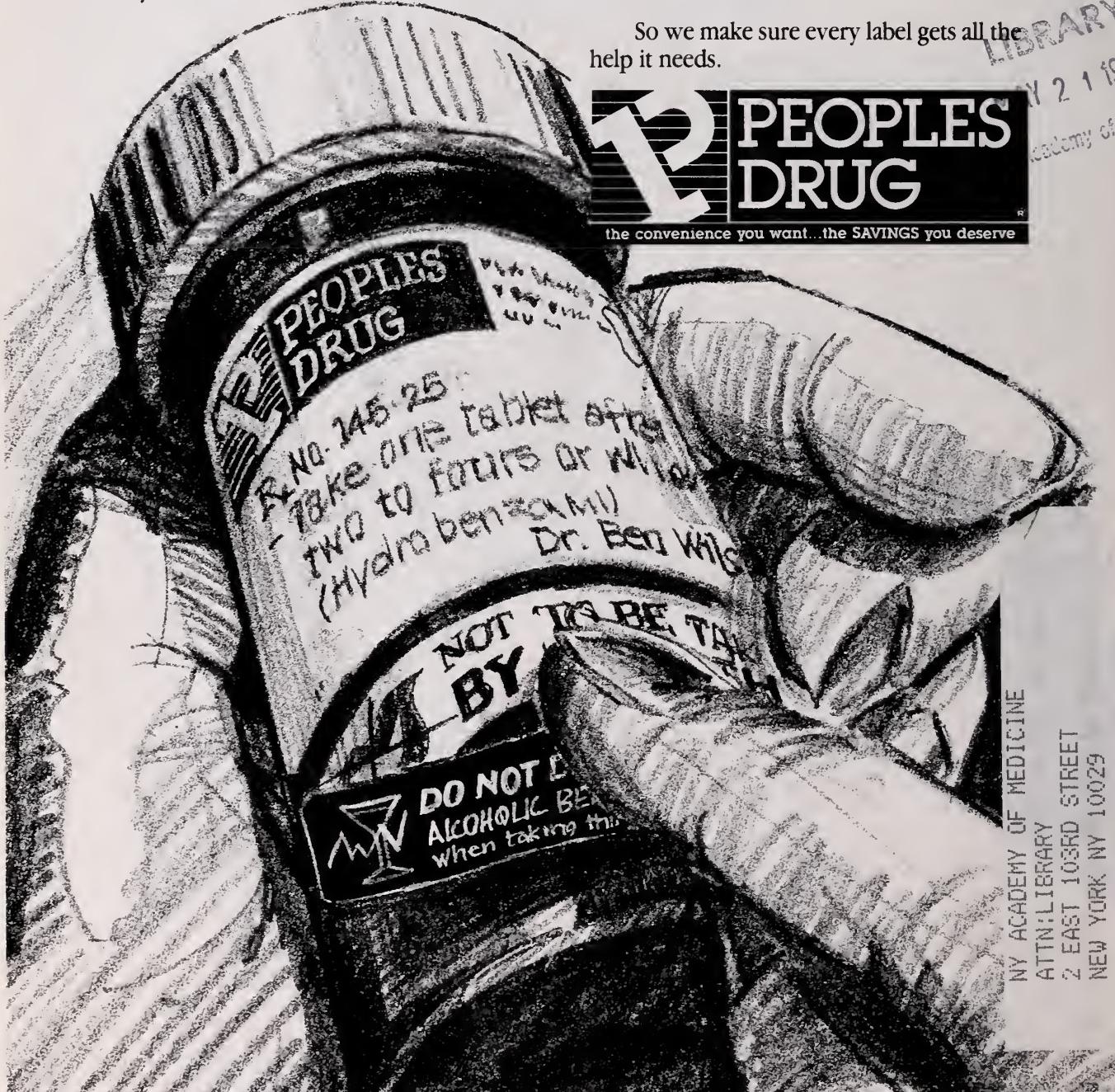
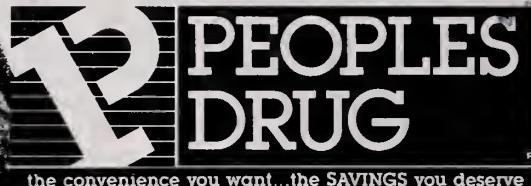
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LETTERS

What info does your laboratory's automated CBC count offer?

Drs. Ende¹ and Becker² are quite correct when they note that *some* automated CBC equipment will fail to pick up significant eosinophilia, basophilia, or left shift in the granulocytic series. However, *some* automated equipment can pick up these abnormalities, or at least alert the operating technologist by noting abnormalities in the shape of the white cell histogram. The proper followup to such an alert is a manual differential. And *other* equipment actually does a cytochemical, flow cytometric differential count, including eosinophils and basophils. And still *other* equipment can perform differential counts by image analysis of Wright stained blood smears. Rarely are total manual eosinophil counts and peroxidase stains necessary in a screening context.

The important message here is that physicians should know the extent of the information available from the automated CBC done in laboratories they utilize. Such information is easily obtained by calling the laboratory director.

R. Lawrence Smith, MD

Virginia Beach General Hospital
1060 First Colonial Road
Virginia Beach VA 23454

1. Ende M. Automated CBC count may obscure L-tryptophan toxicity diagnosis (letter). Va Med 1990;117:141
2. Becker DM et al. Eosinophilia and myalgia associated with L-tryptophan use: case report. Va Med 1990;117:68-70

Encourages reports of complications

Shown below are major complications encountered in patients at the Virginia Heart Institute from March 1973-December 1989. All studies utilized the Sones technique for coronary outpatient arteriography.

Complication	Number	VHI	National (100 Hospitals)
None	1821	.97.6	.97.0
Vascular	30	.1.6	2.0-4.0
Arrhythmia	10	.5	.4
Death	2	.1	.1
Contrast	2	.1	.2
Heart attack	1	.1	.1
Stroke	0	.0	.1
Hemorrhage	0	.0	.1

I would like to encourage other physicians and hospitals to voluntarily list complications of this and other procedures for purposes of quality assurance.

Charles L. Baird, Jr., MD

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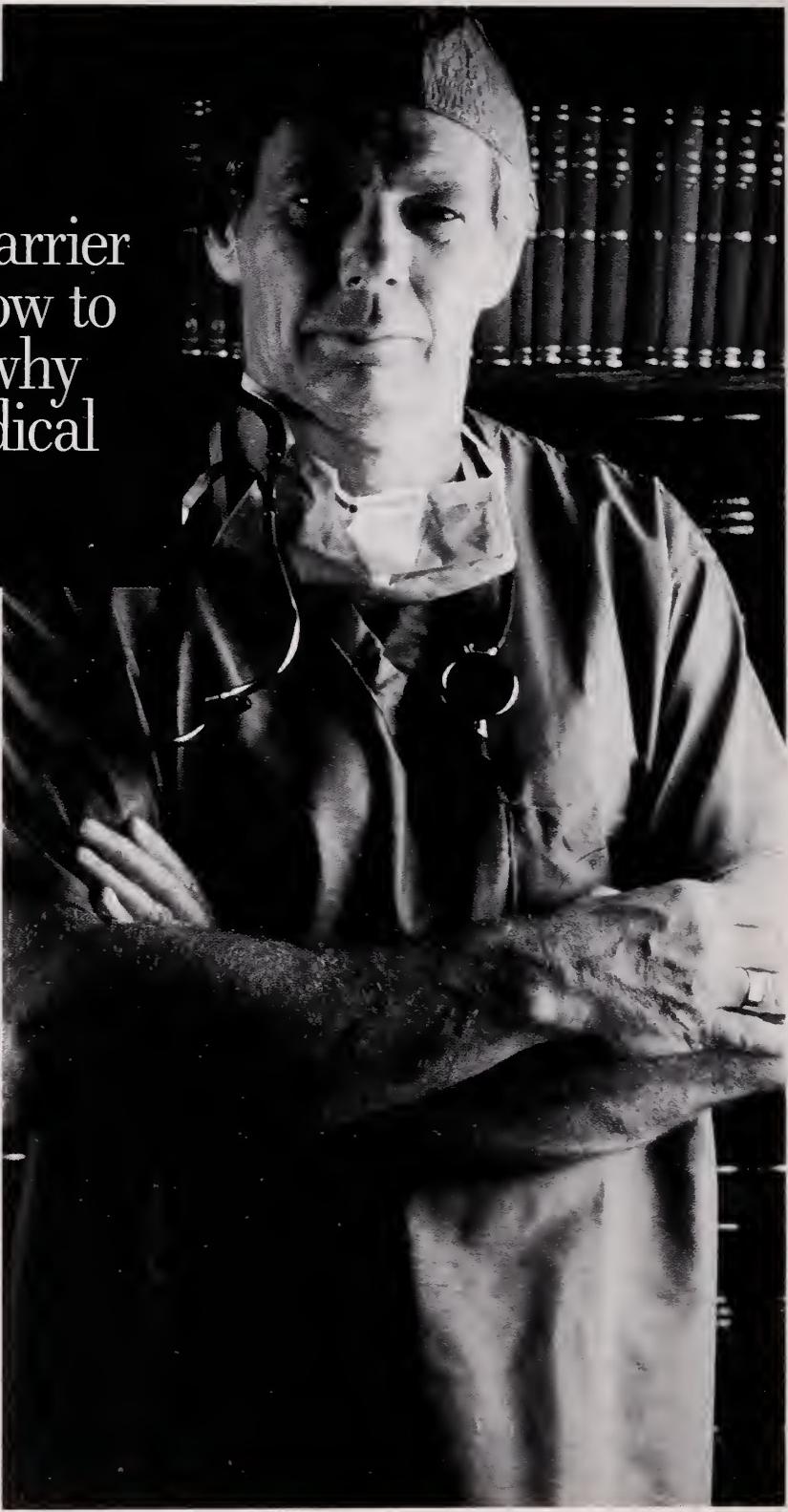
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Injured Infant Act Changes; Bulala Case and the Cap

The following articles appeared originally in the April 1990 edition of The Virginia Section News, a publication of the American College of Obstetrics and Gynecology and the Virginia Obstetrical and Gynecological Society and are reprinted here by permission.

THE 1990 Session of the General Assembly passed three bills that amended the Injured Infant Act. One bill redefines a birth-related neurological injury in the manner requested by the Medical Society of Virginia. The amended statute defines such an injury as one that renders the infant permanently "motorically disabled and (i) developmentally disabled or (ii), for infants sufficiently developed to be cognitively evaluated, cognitively disabled. In order to constitute a 'birth-related neurological injury' within the meaning of the Act, such disability shall cause the infant to be permanently in need of assistance in all activities of daily living. This definition shall apply to live births only and shall not include disability or death caused by genetic or congenital abnormality, degenerative neurological disease, or maternal substance abuse." The revised definition is designed to clarify rather than expand the type of infants protected by the fund and is the product of extensive deliberations by a panel of experts assembled by the Medical Society of Virginia.

The General Assembly also adopted legislation to mandate that the Commissioner of Insurance must require insurance companies writing malpractice insurance in

Virginia to include an appropriate discount from the standard premium if the physician is a participating physician under the Injured Infant Act.

Finally, the General Assembly adopted legislation addressing the circumstances under which an injured infant's exclusive remedy is to the Injured Infant Fund. Currently, in order for an injured infant to obtain compensation from the Fund for a birth-related neurological injury, both the physician and the hospital must participate in the Injured Infant Program. If the physician and the hospital are participants, the exclusive remedy available to an injured infant, his personal representatives, parents, dependents or next of kin on account of such injury is to file a claim for compensation under the Act. The injured infant is precluded from bringing a civil suit against the physician or hospital except where there is clear and convincing evidence that the physician or hospital intentionally or willfully caused or intended to cause a birth-related neurological injury and suit on such claim is filed before the award under the Act becomes final and binding.

The amendments to the Act provide that an infant may obtain compensation from the Fund for a birth-related neurological injury if either the physician or the hospital is a participant in the Program. The Act, as amended, precludes the infant, his personal representatives, parents, dependents or next of kin from bringing a civil suit against a participating physician or hospital (except in the case of intentionally

or willfully caused injuries), but permits the bringing of a civil suit against a nonparticipating physician or hospital as long as no participating physician or hospital is made a party to such suit. However, the commencement of a civil suit against a nonparticipating physician or hospital, regardless of the outcome of the suit, constitutes an election of remedies by the plaintiff and precludes the plaintiff from filing a claim for compensation against the Fund. The amendments also provide that if a claimant elects to file a claim against the Fund and is awarded compensation from the Fund, he loses the right to bring a civil suit against the nonparticipating hospital or physician, but the Fund is subrogated to all of the common law rights based on negligence or malpractice that the claimant had against the nonparticipating physician or hospital.

This portion of the amended Act has two advantages over the current law. First, by electing to participate in the Program a physician can protect himself from civil suits regardless of whether the hospital elects to participate in the Program. Second, severely injured infants are more likely to be protected by the Fund since compensation will be available if either the physician or the hospital is a participant in the Program. A physician or hospital choosing not to participate in the Program remains vulnerable to a civil suit not only by an injured infant but also by the Fund.

All of the foregoing changes become effective on July 1, 1990.

ALLEN C. GOOLSBY, Counsel

ON March 2, 1990, the Virginia Supreme Court released its opinion in the *Bulala v Boyd* case responding to questions of law certified to that Court by the United States Court of Appeals for the Fourth Circuit following the Fourth Circuit's 1989

decision upholding the constitutionality of the Virginia medical malpractice cap on damages. The defendant in this case was an obstetrician/gynecologist who was sued for injuries to an infant, which her mother and her father claimed were created by oxygen deprivation prior to birth.

The case was originally tried to a jury which returned six separate verdicts against Dr. Bulala. These included two verdicts for the child (compensatory damages and punitive damages), two verdicts for the mother (compensatory damages and punitive damages), one verdict for the father (compensatory damages), and one verdict for the father and mother jointly (medical expenses for the child until age 18).

One of the questions certified to the Virginia Supreme Court involved whether, and if so how, the cap is to be applied to multiple plaintiffs and multiple claims by plaintiffs. The Court concluded that the total damages recoverable for injury to a "patient" is limited to the statutory cap, regardless of the number of claims made by that "patient." However, it then decided that, in this case, both mother and the child were to be considered "patients" within the meaning of the Act. The mother was entitled to damages up to the statutory cap for her own physical injury and also her "mental suffering resulting from the birth of a defective child." The Court concluded that at the moment of birth, the child became a "natural person" and a patient of the obstetrician/gynecologist. Accordingly, the child was also entitled to damages up to the statutory cap. In this case, the Court ruled that the father's claim for emotional distress was entirely derivative of the child's claim and was not entitled to a separate statutory cap. Thus in this case, because the child's damages exceeded the statutory cap, the father was not entitled

to a recovery.

Another question certified by the Fourth Circuit was whether the statutory cap applies to an award of punitive damages for acts of medical malpractice. The Court ruled that punitive damages are subject to the cap. In this case, no punishes were recoverable since the amount of the statutory cap had already been met.

The Fourth Circuit also certified to the Virginia Supreme Court the question of whether Virginia law allows recovery for the loss of enjoyment of life in a medical malpractice action. The Supreme Court held that "loss of enjoyment of life" would be duplicative since it is already covered by an award of pain and suffering. Thus damages were not recoverable for a separate

award of "loss of enjoyment of life."

Justices Russell and Poff dissented from the Court's opinion regarding the application of the term "patient" to the child in this case, under the facts presented. They noted that all of the injuries alleged to the child occurred before she was born. Thus, Justice Russell and Justice Poff considered the child's claims as entirely derivative of the mother's claim. Under this interpretation, all injuries to all three plaintiffs "flow from a single 'injury to . . . a patient,' Dr. Bulala's neglect of Helen Boyd [the mother]," making the total amount recoverable \$750,000 (the amount of the medical malpractice cap at the time of this case).

SANDRA L. KRAMER, Counsel

No Dumping Allowed

New Rules for Patient Transfer

Virginia's hospitals are gearing up for the inception on July 1 of new federal strictures relating to the transferring of patients, or, as some would have it, "patient dumping." The new provisions are amendments, passed by Congress last year, to the Consolidated Omnibus Reconciliation Budget Act (COBRA), which was passed by Congress in 1985 and amended previously in 1987.

Since physicians are inextricably involved in patient transfers, Virginia's hospital administrators will be at pains to get fulsome information on the new amendments to their medical staffs. To reinforce those efforts, Va Med offers here, in brief, the salient new provisions relating to physicians. The information is adapted from an article written by Sheila McClanahan, JD, and published in *The Virginia Insurance Reciprocal News* for Jan/Feb 1990.

It should be emphasized first of all that this legislation applies to *all* patient transfers, not just those arising in the emergency room.

The ER setting, however, figures largely in the amendments. For instance, the original statute required medical screenings of a patient presenting to an emergency department in "a medical emergency condition or active labor." As of July 1, 1990, the phrase "or active labor" is out, and *any* patient presenting to an ER must be medically screened to assess whether or not the patient is in a emergency condition.

Note well, however, that the term "emergency condition" is newly defined to include a pregnant woman who is having contractions and 1) there is inadequate time to

effect a safe transfer before delivery and/or 2) a transfer would pose a threat to the health and safety of the woman or her unborn child.

Notable also is the new stipulation prohibiting hospitals from delaying appropriate medical screening and treatment in order to inquire about a patient's method of payment or insurance status.

Generally, the transfer of unstable patients has been forbidden under the Act but with certain exceptions, and the amendments expand on those exceptions, as follows: An unstable patient may be transferred after informing the patient (or one acting on behalf of the patient) of the hospital's obligations and the risks of transfer, and after obtaining the individual's written request for transfer, accompanied by a certification contemporaneously signed by a physician that the medical benefits reasonably expected at another medical facility outweigh the risk to the patient (and, in the case of labor, to the unborn child) as a result of the transfer, or a signed certification by a qualified medical person in a physician's absence. This person must consult with a physician and certify the transfer at the direction of the consulting physician, who must co-sign the document. Note: "Qualified medical person" is not defined by the regulations.

What if a physician refuses to transfer a patient with an unstable emergency medical condition? As of July 1, 1990, the amendments absolve the refusing physician from any penalty, nor may the hospital take any adverse action against the physician for this refusal.

Also expanded under the new amendments is the content of the medical record that must accompany the transferring patient to the receiving facility. It must now include all of the following: records related to patient's emergency room condition; observations of

signs/symptoms; preliminary diagnosis; treatment provided; results of any tests; the informed written consent or certification (including the name and address of any on-call physician who has refused or failed to appear within a reasonable time to provide necessary stabilizing treatment).

The amendments define the "responsible physician" subject to the Act's penalties. He or she is "one employed by, or under contract with, the participating hospital and acting as an employee or under such contract, having professional responsibility for the provision of examination or treatment for the individual or transfer of the individual." This physician is subject to a civil fine of not more than \$50,000 per violation and/or temporary or permanent suspension of his/her Medicare providers agreement.

Also subject to these penalties as of July 1, 1990, will be a physician who is "responsible for the examination, treatment, or transfer of an individual in the participating hospital, including the physician on call, who knowingly violates a requirement of COBRA, and/or 1) signs a certification when the physician knew or should have known that the benefits did not outweigh the risks; or 2) misrepresents an individual's condition or other information, including a hospital's obligation under the Act."

Rendered immune from the penalties, however, is the initial treating physician who determines that the patient requires the services of one of the hospital's on-call physicians and who so notifies the on-call physician, but the on-call physician fails or refuses to respond "within a reasonable time." In these circumstances, the initial treating physician may order a transfer without incurring a civil penalty or fine. The hospital and/or the dilatory on-call physician, however, may be in trouble.

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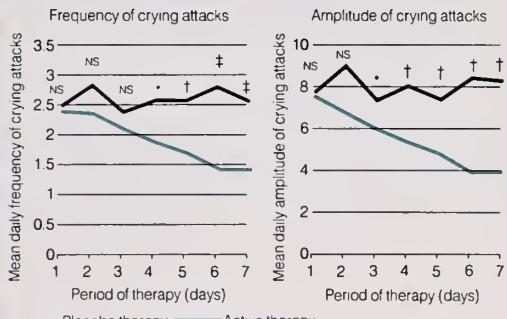
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1. Kanwaljit SS, Jasbir KS. Simethicone in the management of infant colic. *Practitioner*. 1988;232:508.

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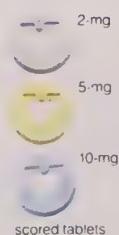
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Captain, U.S. Army Reserve.

EDUCATION University of Southern California, B.S.;
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RESIDENCY Harbor General Hospital—UCLA
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HOSPITAL AFFILIATIONS St. Luke Hospital;
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References

1. USP DI Update, September/October 1988, p 120.
2. Br J Clin Pharmacol 1985;20:710-713.
3. Data on file, Lilly Research Laboratories.
4. Scand J Gastroenterol 1987;22(suppl 136):61-70.
5. Am J Gastroenterol 1989;84:769-774.

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Carcinogenesis, Mutagenesis, Impairment of Fertility—A two-year oral carcinogenicity study in rats with doses as high as 500 mg/kg/day (about 80 times the recommended daily therapeutic dose) showed no evidence of a carcinogenic effect. There was a dose-related increase in the density of enterochromaffin-like (ECL) cells in the gastric oxyntic mucosa. In a two-year study in mice, there was no evidence of a carcinogenic effect in male mice, although hyperplastic nodules of the liver were increased in the high-dose males as compared with placebo. Female mice given the high dose of Axid (2,000 mg/kg/day, about 330 times the human dose) showed marginally statistically significant increases in hepatic carcinoma and hepatic nodular hyperplasia with no numerical increase seen in any of the other dose groups. The rate of hepatic carcinoma in the high-dose animals was within the historical control limits seen for the strain of mice used. The female mice were given a dose larger than the maximum tolerated dose, as indicated by excessive (30%) weight decrement as compared with concurrent controls and evidence of mild liver injury (transaminase elevations). The occurrence of a marginal finding at high dose only in animals given

an excessive and somewhat hepatotoxic dose, with no evidence of a carcinogenic effect in rats, male mice, and female mice (given up to 360 mg/kg/day, about 60 times the human dose), and a negative mutagenicity battery are not considered evidence of a carcinogenic potential for Axid.

Axid was not mutagenic in a battery of tests performed to evaluate its potential genetic toxicity, including bacterial mutation tests, unscheduled DNA synthesis, sister chromatid exchange, mouse lymphoma assay, chromosome aberration tests, and a micronucleus test.

In a two-generation, perinatal and postnatal fertility study in rats, doses of nizatidine up to 650 mg/kg/day produced no adverse effects on the reproductive performance of parental animals or their progeny.

Pregnancy—Teratogenic Effects—Pregnancy Category C—Oral reproduction studies in rats at doses up to 300 times the human dose and in Dutch Belted rabbits at doses up to 55 times the human dose revealed no evidence of impaired fertility or teratogenic effect; but, at a dose equivalent to 300 times the human dose, treated rabbits had abortions, decreased number of live fetuses, and depressed fetal weights. On intravenous administration to pregnant New Zealand White rabbits, nizatidine at 20 mg/kg produced cardiac enlargement, coarctation of the aortic arch, and cutaneous edema in one fetus, and at 50 mg/kg, it produced ventricular anomaly, distended abdomen, spina bifida, hydrocephaly, and enlarged heart in one fetus. There are, however, no adequate and well-controlled studies in pregnant women. It is also not known whether nizatidine can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Nizatidine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers—Studies in lactating women have shown that 0.1% of an oral dose is secreted in human milk in proportion to plasma concentrations. Because of growth depression in pups reared by treated lactating rats, a decision should be made whether to discontinue nursing or the drug, taking into account the importance of the drug to the mother.

Pediatric Use—Safety and effectiveness in children have not been established.

Use in Elderly Patients—Healing rates in elderly patients were similar to those in younger age groups as were the rates of adverse events and laboratory test abnormalities. Age alone may not be an important factor in the disposition of nizatidine. Elderly patients may have reduced renal function.

Adverse Reactions: Clinical trials of varying durations included almost 5,000 patients. Among the more common adverse events in domestic placebo-controlled trials of over 1,900 nizatidine patients and over 1,300 on placebo, sweating (1% vs 0.2%), urticaria (0.5% vs <0.01%), and somnolence (2.4% vs 1.3%) were significantly more common with nizatidine. It was not possible to determine whether a variety of less common events was due to the drug.

Hepatic—Hepatocellular injury (elevated liver enzyme tests or alkaline phosphatase) possibly or probably related to nizatidine occurred in some patients. In some cases, there was marked elevation (>500 IU/L) in SGOT or SGPT and, in a single instance, SGPT was >2,000 IU/L. The incidence of elevated liver enzymes overall and elevations of up to three times the upper limit of normal, however, did not significantly differ from that in placebo patients. Hepatitis and jaundice have been reported. All abnormalities were reversible after discontinuation of Axid.

Cardiovascular—In clinical pharmacology studies, short episodes of asymptomatic ventricular tachycardia occurred in two individuals administered Axid and in three untreated subjects.

CNS—Rare cases of reversible mental confusion have been reported.

Endocrine—Clinical pharmacology studies and controlled clinical trials showed no evidence of antiandrogenic activity due to nizatidine. Impotence and decreased libido were reported with equal frequency by patients on nizatidine and those on placebo. Gynecomastia has been reported rarely.

Hematologic—Fatal thrombocytopenia was reported in a patient treated with nizatidine and another H₂-receptor antagonist. This patient had previously experienced thrombocytopenia while taking other drugs. Rare cases of thrombocytopenic purpura have been reported.

Integumentary—Sweating and urticaria were reported significantly more frequently in nizatidine- than in placebo-treated patients. Rash and exfoliative dermatitis were also reported.

Hypersensitivity—As with other H₂-receptor antagonists, rare cases of anaphylaxis following nizatidine administration have been reported. Because cross-sensitivity among this class has been observed, H₂-receptor antagonists should not be administered to those with a history of hypersensitivity to these agents. Rare episodes of hypersensitivity reactions (eg, bronchospasm, laryngeal edema, rash, and eosinophilia) have been reported.

Other—Hyperuricemia unassociated with gout or nephrolithiasis was reported. Eosinophilia, fever, and nausea related to nizatidine have been reported.

Overdosage: Overdoses of Axid have been reported rarely. If overdosage occurs, activated charcoal, emesis, or lavage should be considered along with clinical monitoring and supportive therapy. Renal dialysis for four to six hours increased plasma clearance by approximately 84%.

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MEETINGS

**1990 Annual Meeting
The Medical Society of Virginia
October 31-November 4**

July 12-14

6th Annual Berkshire Medical Conference: Advances in Cardiology (University of Massachusetts), *Hancock, Massachusetts*. Berkshire AHEC, 413-447-2417.

July 13-15

Workshops in Traumatic Brain Injury Rehabilitation (Medical College of Virginia/VCU). *Virginia Beach*. CME Office, 804-786-0494.

July 16-20

Control of Biohazards in the Research Laboratory (Johns Hopkins), *Baltimore*. Byron S. Tepper, PhD, 301-955-5918, or Dr. Jacqueline Corn, 301-955-2609.

July 18-22

13th Annual Flap Dissection Workshop (Eastern Virginia

Medical School), *Virginia Beach*. CME Office, 804-446-6140.

July 19-21

6th Annual Berkshire Medical Conference: Special Challenges in General Medicine (University of Massachusetts). *Hancock, Massachusetts*. Berkshire AHEC, 413-447-2417.

July 20-22

Practical Internal Medicine (Medical College of Virginia/VCU), *Virginia Beach*. CME Office, 804-786-0494.

July 26-29

40th Annual Scientific Assembly of the Virginia Academy of Family Physicians, Cavalier Hotel, *Virginia Beach*. 21½ credit hrs. VAFP headquarters, 804-358-1721.

July 27-29

12th Annual Pediatric Primary Care Conference: Pediatrics at the Beach (Medical College of Virginia/VCU), *Virginia Beach*. CME office, 804-786-0494.

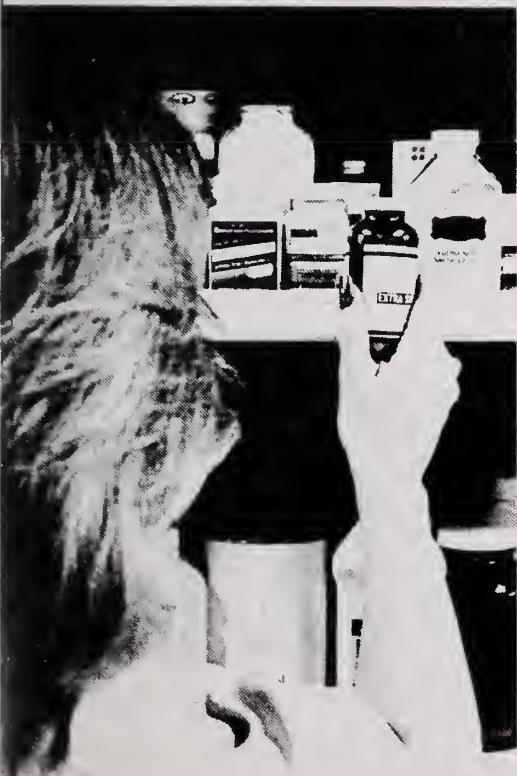
August 10-12

Diabetes and Vascular Disease (Eastern Virginia Medical School), *Virginia Beach*. CME Office, 804-446-6140.

Continued next page

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MORE MEETINGS

August 24-27

12th Edition, Practical Dermatology for the Primary Care Physician (Eastern Virginia Medical School), *Orlando, Florida.* CME Office, 804-446-6140.

September 8

Regular meeting of the Medical Society of Virginia's Council, *Richmond.* James L. Moore, Jr., 804-353-2721.

September 7-9

Annual Meeting/Scientific Session of the Virginia Vascular Society, The Homestead, *Hot Springs.* Dr. Jesse Davidson, program chairman, 703-345-1561.

September 10-14

Basic Course in Nosocomial Infection Control (University of Virginia). *Charlottesville.* Debbie Crickenberger, 804-924-2777.

September 28

13th Annual Perinatal Conference (Medical College of Virginia/VCU). *Richmond.* CME Office, 804-786-0494.

October 3-5

Recent Advances in Clinical Medicine (University of Virginia), Omni Hotel, *Charlottesville.* Lauvonda Young, 804-924-2090.

October 3-6

16th Annual Topics in Gastroenterology and Liver Disease (Johns Hopkins), *Baltimore.* 23.5 credit hrs. Physician fee: \$425. CME Office, 301-955-2959.

October 4-6

Virginia Occupational Health Conference (Medical College of Virginia/VCU), *Richmond.* CME Office, 804-786-0494.

October 8-11

13th Annual Postgraduate Course in Radiology (University of Virginia), Boar's Head Inn, *Charlottesville.* 20 credit hrs. Fee: \$400. Dr. Theodore E. Keats, 804-924-2781.

October 8-12

Basic Course in Nosocomial Infection Control (University of Virginia), *Charlottesville.* Debbie Crickenberger, 804-924-2777.

October 22-27

32nd Annual Emil Novak Memorial Course: Gynecology, Gynecological Pathology, Endocrinology, and High-Risk Obstetrics (Johns Hopkins), *Baltimore.* 44.5 credit hrs. Physician fee: \$650. CME Office, 301-955-2959.

November 3

Symposium on Current Controversies in Colon and Rectal Cancer (University of North Carolina at Chapel Hill), *Research Triangle Park, North Carolina.* CME Office, 919-962-2118.



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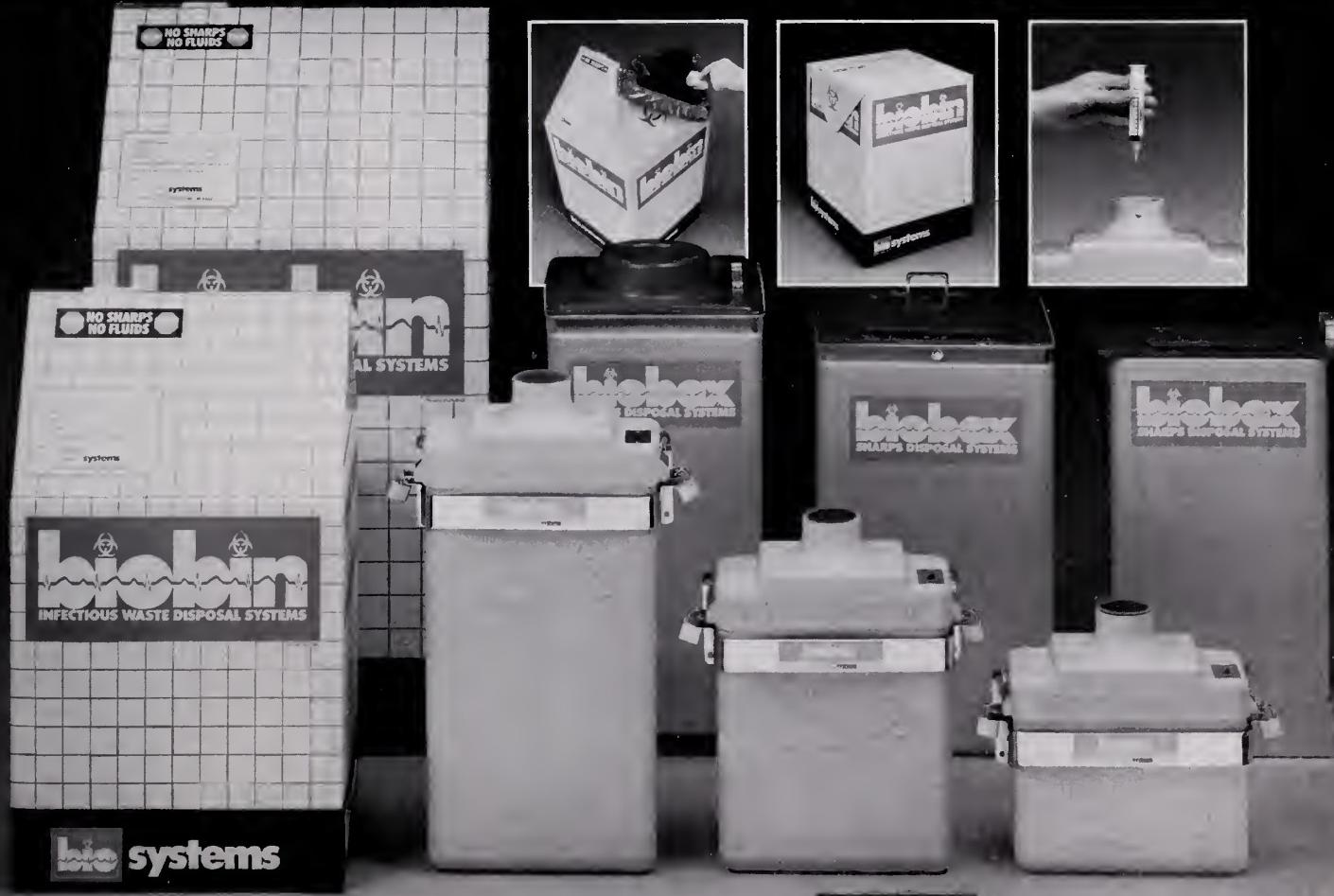
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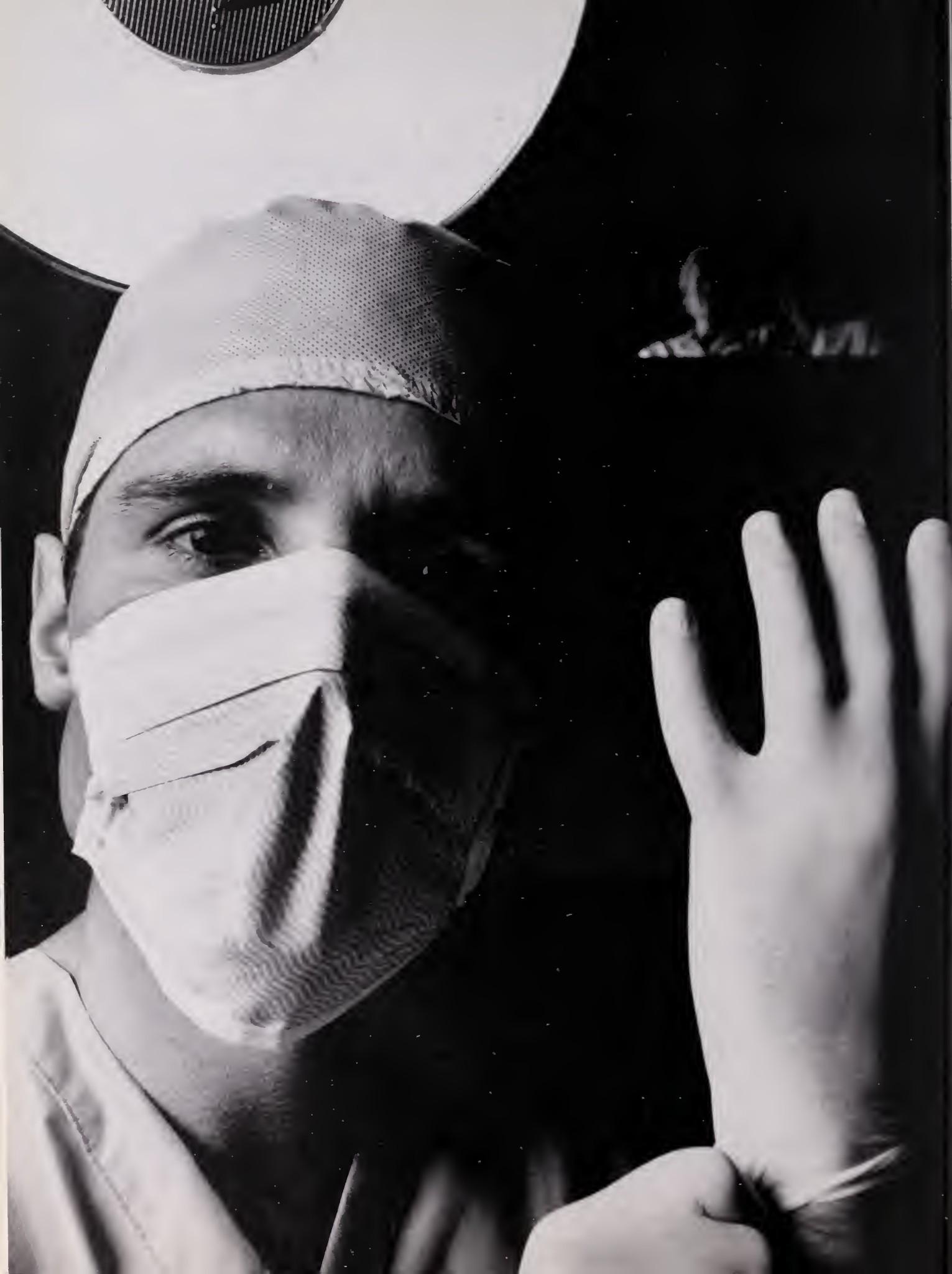


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The Auxiliary and the Teen-Ager

Notes and photographs from a big production starring healthy behavior

From all over Virginia the teen-agers and their teachers came, almost one thousand of them. A March snowstorm had slowed the yellow school buses, and the late-arriving youngsters made a beeline for the shelter of the Convention Centre in downtown Richmond. Their target was the Teen Health Forum, an all-day event organized by the Medical Society of Virginia Auxiliary to promote healthy behavior in Virginia's youth.

The Centre's big auditorium was

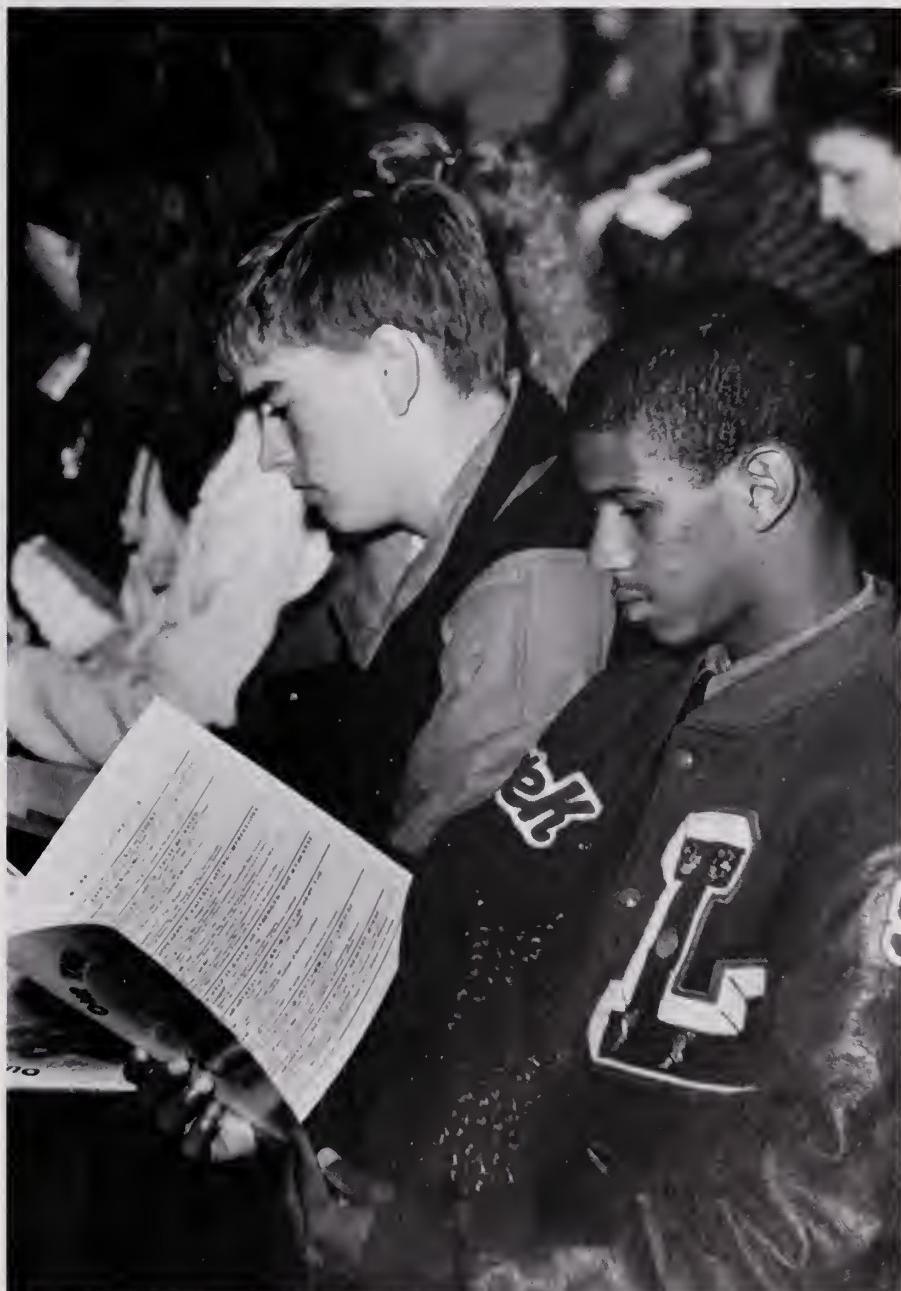


volunteers from the audience, for example, he put on little playlets illustrating ways to combat the peer pressure of drug users. Next Miss Virginia, Tami Elliott, accompanied by high-decible music, wrapped up the audience by bounding and flipping all over the stage in a high-energy gymnastic exhibition.

At mid-morning the teen-agers adjourned en masse for the serious stuff, the breakout sessions. At the other end of the Centre and up one flight eleven classrooms were in readiness. Eleven presenters, most of them physicians, were ready, too, to lead discussions on such

topics as sexuality, drug abuse, emotional disorders, self-image, fitness and exercise. Which to choose, that was the question. Postponing teen-age sexuality? Eating disorders? The perfect diet? Sexually transmitted diseases/AIDS? Steroid abuse? Tobacco abuse? Date rape? Teen suicide? Teen pregnancy? Exercising safely? Driving safely? Finally everyone was settled in the session of choice—but for the afternoon sessions, everyone had to choose all over again.

At noon there were 1,000 box lunches for the taking, and students



the youngsters' home room for this Tuesday learning experience. From the stage Mrs. Carroll T. Petty and Mrs. Phillip Thomason, MSVA president and vice president, and Dr. William H. Barney, Medical Society of Virginia president, welcomed the youngsters. They were followed by the keynote speaker, Mike Green, once an all-star high school athlete and now a recovering alcoholic who has been off the booze for twelve years. He looked young, he talked young, and he had showmanship; with

and teachers picnicked together in the Centre's hallways. Then came another galvanizing interval in the auditorium, where students of the Virginia Karate Society performed dramatic feats of self-defense and the heavy beat of the House Patrol, a rapper and dancers band, vibrated compellingly through the hall. The winners of the essay contest ("Our Youth—Our Future") stepped up to the stage to receive from their peers a big hand and from Mrs. Petty some big checks (\$500, \$250, \$125). Late in the day, as a going-away present, all teen-agers and teachers received T-shirts imprinted with the Teen Health Forum's logo.

It was a gigantic production, and the persons who made it go, from start to finish, were Vivian Petty and Eileen Thomason.

Two other prime movers and shakers were S. John Davis, PhD, superintendent of public instruction for Virginia's schools, and Jeane L. Bentley, of the State Department of Education.

Key roles were played by these physicians who worked up and conducted breakout sessions: Dr. Michael E. Bohan, Portsmouth;

Photographed at right, Dr. Greg Leather's breakout session on exercising safely.

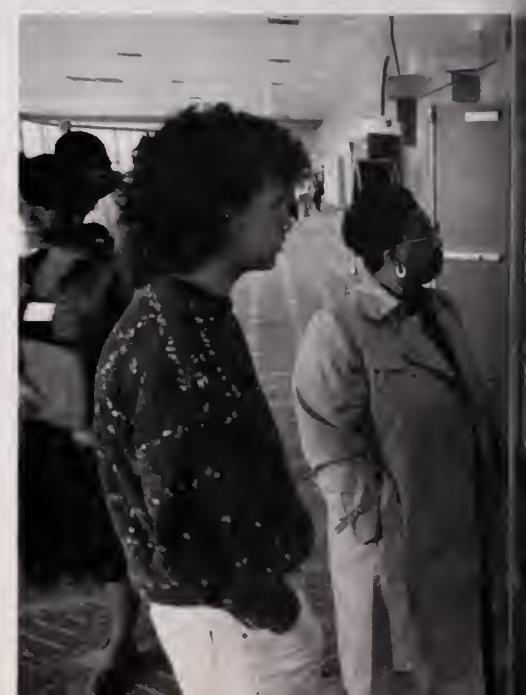
Below left, Miss Virginia onstage. Below center, Dr. Warren Klam in hallway with students and teacher after his session on drug abuse. Far right below, Dr. Michael Bohan's session on steroids.

Dr. David R. Faber II, Norfolk; Dr. Warren P. Klam, Falls Church; Dr. Greg Leather, Arlington; and from Richmond, Dr. Lawrence E. Blanchard III, Dr. Kevin R. Cooper, Dr. Hazel S. Konerding, Dr. Dean X. Parmalee, Dr. Harold F. Young, and Dr. Joseph R. Zanga.

And these groups contributed that vital ingredient, money: Norfolk Academy of Medicine Auxiliary; Portsmouth Academy of Medicine Auxiliary; Portsmouth General Hospital Foundation Fund; Maryview Medical Center; Johnston-Willis Hospital; St. John's Hospital; Chippenham Hospital; St. Mary's Hospital; Allen, Allen, Allen & Allen; Virginia Insurance Reciprocal; and Southern Health.

—A.G.

Photographs by Taylor Dabney





Legalizing Drugs: A Few Simple Questions

As time passes, more and more people are suggesting that recreational street drugs be legalized. Aside from a few libertarians, most people offer the rationale that decriminalizing usage will remove the profit motive and therefore the presence of drug pushers. They seem to be saying that if you can't lick them, join them, and at the same time unclog the legal and penal systems; however, details about what will take the place of the present system are vague, and perhaps a few simple questions are in order.

The first question to be asked is, who will be selling the drugs, the private market place or the government? The former is less likely, but if the private sector is given the task, will it be on a monopoly basis or on the basis of competition within the free enterprise system? If a monopoly, who will get it and how will it be regulated? If regulated, by whom? How will prices be set? Will it be regulated like a utility or will the sellers be free to charge what the market will bear? Which drugs will be sold and will the sellers be free to introduce new drugs on their own? If competition is the order of the day, will advertising be permitted, at least to the extent that tobacco and alcohol products are permitted to advertise? If not, and it is legal, why not? Will "specials" be allowed to be offered to increase the user population? Will new "designer" drugs be developed and marketed? Will exports be permitted, to improve our balance of payments?

If, as is more likely, the government takes on the task, will it be in the hands of the local, state or federal government? If either of the former, won't chaos result from different regulations, different drugs permitted to be sold, and at different prices? Since that is likely to happen, it would fall on the federal government to sell the drugs out of "state stores" as some states now sell liquor. Now a host of new questions arise. Would the government advertise? Would it sell all drugs or just some, leaving the market for the others in the hands of drug dealers? Would it take the lead in introducing new drugs, such as the "ice" coming in from Japan, or would it leave that to the dealers? Would it set prices high to discourage use and therefore open the market to "moonshiners," who would be able to sell cheaper because of less fixed costs (one can imagine the size of the bureaucracy it would take to set up such a program), or would it set the price low and thereby encourage broader usage?

Would it be restrictive in its sales practices, trying to discourage abuse (and thereby making it easier to go to your neighborhood pusher), or would it be lenient, thereby encouraging broader usage? Obviously, it would not sell to minors, but does anyone familiar with teen-agers doubt their ability to obtain the drugs one way or the other? If not sold to minors, would this not leave this entire market open to drug dealers? In the name of "fairness" would the government distribute the drugs free of charge to indigents? Will this become a basic right discovered somewhere in the Constitution? Not if, but when side effects occur, will it be the responsibility of the government, since it is doing the selling, to provide appropriate care? Will this open the door to a whole new class of lawsuits? If an addicted or deformed or impaired baby is born, will it be the responsibility of the government to care for it? Forever?

If it is decided to distribute the drugs through clinics run by physicians, new questions arise. Again, will they be limited to some drugs or to all? Will it be limited to addicts or to anyone who expresses an interest in them? If the former, will not the recreational drug users, who are in the majority, continue to get their supplies from dealers? With addicts, who decides how much is enough to satisfy their needs? If the clinic staff decides, will not many addicts take the pure drug, adulterate it, and become dealers themselves, so that they have money to meet their needs, as they and not the clinic determine it? Will addicts be forced to undergo treatment, and if so, is it a violation of their rights as some people would undoubtedly see it? What will be considered an acceptable recidivism rate when requests come to the Congress or the state legislatures for refunding? What would be the total costs of such a program?

Currently those who smoke can do so in smoking lounges or sections of restaurants designated for smokers, while those who wish to imbibe alcohol in convivial company and surroundings can do so in a wide variety of bars, taverns, restaurants, clubs, etc. Is there any reason, with recreational drugs legalized, why there should not be similar places for those who wish to do the same with drugs? Could we deny the opening of shooting galleries for heroin, crack and coke bars and the like? Perhaps we could even have a TV sitcom based on the friendly camaraderie which

Cocaine: Recognizing, Treating the Abuser

Peter Coleman, MD
Richmond, Virginia

would exist in one of these establishments.

If recreational drugs are legalized, why should we keep intact the elaborate system to supervise the dispensing of controlled substances—narcotics, tranquilizers and the like? Why should we not sell morphine and codeine and their derivatives over the counter, next to the aspirin, acetaminophen and ibuprofen? Why should we not sell major tranquilizers and sedatives the same way? Why not all prescription drugs?

A further concern is the educational system we have painstakingly put in place over the past two decades against abuse of alcohol, tobacco products and recreational drugs. How seriously will anyone take these campaigns when the government passively (in effect actively) endorses the use of recreational drugs? Virtually everyone across the political spectrum agrees that a fundamental role of government is to maintain public safety. What amount of testing will be necessary to protect us from harm? We immediately think of airline pilots, air traffic controllers, railroad engineers and tanker captains, but would not everyone who makes decisions which affect our health and welfare be subject to testing? Would this not include all vehicle drivers? What size police and support system would be necessary to screen the roads for drug use in addition to alcohol use? We are aware of the increasing competitiveness of the world economy. Is legalization of drugs likely to improve or decrease our productivity and competitiveness?

There are obviously many more questions to be asked and the ones asked above will undoubtedly raise many more related ones; however, the greatest questions relate to our ability, and will, to deal with difficult and dangerous problems. Is our solution to such a difficult and intractable problem to give in to it, or to devise effective ways of fighting it? If this problem is difficult, aren't many of our other social problems? Should we give in to them as we give in to this? The answer depends on where we are going as a society.

DAVID A. ZOHN, MD

Dr. Zohn is in the private practice of physical medicine and rehabilitation at 1515 Chain Bridge Road, Suite 102, McLean VA 22101.

COCAINE is taking a severe toll of the people and industry of this nation. It has become increasingly linked with new AIDS cases, with sky-rocketing crime and murder rates in many of our cities, and there are increasing numbers of cocaine-addicted pregnant mothers. Cocaine-related deaths in the United States increased by over 300% between 1982 and 1985.¹ While there is some evidence that attitudes about cocaine are beginning to improve and that use of cocaine is beginning to decline in upper-middle class sectors of our society, there is no end in sight for the devastation that this drug is producing.

History and Epidemiology

Cocaine is found in the leaves of the coca plant (no relation to the cocoa plant, from which chocolate is made). The coca plant is indigenous to many countries in South America. The effects of the plant have been known since around 500 AD but it was not until the late 1800s that the active drug was isolated and its effects described. There followed a small epidemic, which seemed to terminate a few years later, partly due to passage of the Harrison Drug Act of 1914. The serious consequences of cocaine addiction were well described at this time. Sigmund Freud was one of the many well-known people who used the drug; he advocated its use to treat alcohol and morphine addiction. Interestingly, it was not too much later that morphine was being tried as a cure for cocaine addiction.² Following this small epidemic, cocaine use remained uncommon and fairly much confined to use by addicts of other drugs, especially heroin. In the late 1960s cocaine use began to increase, initially glamorized by the media and the elite of society. Until 1980 it was perceived as a non-addictive and harmless drug with as few problems as marijuana. Even the medical profession regarded cocaine as relatively harmless. In 1980 the official manual of the American Psychiatric Association, the DSM III, did not recognize cocaine as having a withdrawal syndrome or producing dependence.³ Use rapidly spread throughout society and

Dr. Coleman conducts a private practice in addictive diseases. Address correspondence to him at 2115 Grove Avenue, Richmond VA 23220.

Submitted 12-28-89.

has now spread across all socioeconomic boundaries.

Pharmacology

Cocaine exists in the plant as a base, and its extraction involves the addition of a strong acid to form cocaine hydrochloride. In this form it is a white crystalline powder which is readily soluble in water but has a very high vaporization point. Cocaine is used in predominantly three ways. 1) Most commonly cocaine hydrochloride is insufflated up the nose so that it can be absorbed through the nasal mucosa. Absorption takes from 1-3 minutes and is probably less than 50% complete because of the intense vasoconstriction that the drug causes. 2) Much less commonly, cocaine hydrochloride can be dissolved in water and injected intravenously. This method is often used for addicts who already abuse heroin, is 100% efficient and the effects begin in only 30-120 seconds. 3) The most rapidly increasing method of use is for cocaine users to smoke the drug as the "free-base." Freebase, or "crack," is produced when the chloride ion is removed from cocaine hydrochloride. This can be done fairly readily at home, but drug dealers have recently learned that they can mass produce the freebase and sell it as the more potent form under the name of crack. Crack is so-called because of the sound it makes when vaporized in a pipe. Crack or freebase has a much lower vaporization point than cocaine hydrochloride and is easily smoked in a pipe. It is rapidly absorbed through the lungs, and because the pulmonary circulation then carries the drug directly to the left ventricle and on to the brain, its effects begin in only 5-10 seconds. It thus produces a very intense high, which leads to an equally intense crash. There follows a very strong desire to repeat the experience and attempt to regain the pleasurable feelings.

Clinical Effects

Cocaine causes an increase in sympathetic tone with vasoconstriction, hypertension and tachycardia. It has been found to cause cerebrovascular accidents, TIAs, myocardial infarction, angina and ruptured ascending aorta.⁷ In addition, it is known to cause seizures and ventricular arrhythmias. If used intravenously it is associated with thrombosis, sepsis, bacterial endocarditis and AIDS. Intranasal use frequently leads to chronic rhinorrhea, inflammation and sometimes a perforated septum. Use during pregnancy, even the first trimester, is associated with spontaneous abortion, placental abruption and stillbirth. The infants may be very irritable, have problems learning, cerebral hemorrhage, neonatal seizures or other birth defects, including genitourinary anomalies and ileal atresias.⁸

In general, people using cocaine report that they feel euphoric, powerful, energetic, and very much in control. They become talkative, excited, self-confident and even grandiose and out of touch with reality.

There seems to be an increase in sexual libido at low doses although at higher doses a decrease in sexual performance develops. There is usually some anxiety caused by cocaine use and this becomes much worse at higher doses. Physically, there is an increase in pulse rate and blood pressure, some muscle contractions occur, and there may be urinary and bowel retention. At toxic levels a marked anxiety develops, as well as a serious loss of judgment and paranoid delusions.

The acute, intense euphoria generally lasts only a few minutes and is replaced by a less intense euphoria with increasing anxiety and signs and symptoms of sympathetic discharge. This period of less intense euphoria lasts about 30-90 minutes and is gradually replaced with increasing feelings of anxiety, dysphoria, distress, depression and drug craving—the cocaine crash. Users report that during this cocaine crash they feel intensely uncomfortable and they will often go to extraordinary lengths to avoid these negative feelings. Many users take large amounts of alcohol or other sedatives to avoid this dysphoria.

Chronically, cocaine use causes a wide variety of problems. Most commonly users begin to feel out of control and depressed. There is a disruption of sleep patterns and appetite. There is a generalized loss of interest in life, or anhedonia. Initially these feelings of depression and anhedonia can be relieved by more cocaine, but most chronic users report that they can never quite recapture the good feelings that cocaine originally produced. Some chronic abusers use the drug on a daily basis, but a more common pattern is of binge use about 2-3 times per week. Abusers will start on a binge, often associated with payday, and then not stop until the supply is exhausted, often 2 or 3 days later. This binge pattern gives the abuser some illusion of control since many abusers believe that if they can go for 3-5 days without using, then they can control their use any time they choose to.

Even though the medical problems associated with cocaine abuse are dramatic, they are reasonably uncommon, and if physicians wait for these problems to arise they will be missing a lot of cocaine abusers who come into the office. It is important, then, for all physicians to consider cocaine abuse with all patients. Usually a straightforward question, as part of a general health appraisal, asking whether cocaine has ever been used will elicit an honest response. If the response is in the affirmative, then a followup question of whether cocaine has ever been a problem will be a useful introduction to talking about the subject.

Neurochemistry

Cocaine exerts its effects on a number of neurotransmitter systems, although the exact details are not completely clear. Animal experiments and human objective and subjective experience seems to point to the dopamine system as being the system most responsi-

ble for cocaine's effects.⁴ Cocaine rapidly blocks the reuptake of dopamine into the presynaptic nerve cleft and thus allows dopamine's action to be intensified and prolonged. There may in addition be increased dopamine synthesis, and there is reason to suspect that there follows an "up-regulation" of dopamine receptors, which refers to the ability of the nervous system to try to achieve a homeostatic state in the face of too much neurotransmitter. The actual number of dopamine receptors seems to increase, probably in an attempt to use up the excess neurotransmitter. Unfortunately, what ultimately occurs is that these increased receptors then seem to "demand" higher levels of dopamine to occupy them, and this probably accounts for some of the severe drug craving that addicts experience once the cocaine is finished. While cocaine makes more dopamine acutely available in the central nervous system, it causes chronic dopamine depletion, as all of the dopamine is kept in the synaptic cleft and therefore rapidly broken down.

Cocaine's stimulation of the dopamine system seems to occur in the limbic system, the basal ganglia and substantia nigra. The limbic system, and predominantly the nucleus accumbens, are intimately connected with the brain's pleasure and reward centers. These areas are strongly associated with the brain's primitive drives for food, water and sex and are generally under less conscious control than other "higher" areas of the brain.

Experiments with animals and in humans reveal that cocaine is intensely addicting physically.^{5,6} Experiments with animals have revealed that animals will prefer cocaine over most other known drugs and over food and water. If left alone with all the cocaine they can use, 90% of animals will be dead in 30 days (as opposed to 36% with heroin). Animals will continue to seek cocaine even if they receive an electric shock paired with the cocaine. Some researchers train animals to self-inject drugs until the animals become addicted to the drug. Once an animal has become addicted to a drug an experiment can attempt to determine how addictive that drug is by monitoring how often the animal will continue to press the lever to try to get more drug, even after the drug supply is turned off. For Valium,[®] the average number of lever presses after the drug supply is turned off is 50 presses, for heroin it is 500 presses, for cocaine the animals will continue to press the lever an average of 12,000 times even after the cocaine supply is turned off. Our experience with human addicts also indicates that the drug is intensely addictive and the cravings powerful.

Treatment

Treatment is best performed in a specialized setting staffed by professionals experienced with drug and alcoholism patients. During the cocaine crash patients usually require little more than supportive therapy. Following the cocaine crash (1-5 days), treatment is

similar to that generally accepted for other drugs of abuse, with some distinct differences. It is generally accepted that it is important for patients to abstain from all mood-altering drugs, including alcohol.⁹

Group therapy is generally felt to be the most effective technique to help the patient realize and accept how serious the cocaine problem has become. The goal of early treatment is to have the patient be able to diagnose himself as having a drug problem, as a means to allowing him to accept fully how serious the problem has become. Helping patients to overcome this denial is very difficult and in practice this process frequently takes a number of weeks. The denial for patients with cocaine abuse problems seems to be a little different from that associated with patients who abuse alcohol. Cocaine addicts seem to accept readily that they are unable to control their use once they start using cocaine; their denial seems to involve a difficulty in accepting how difficult it will be to avoid returning to the drug even after they have gone through withdrawal and become drug-free.

Once the patient has accepted the nature and severity of the problem, he or she needs to learn and practice relapse prevention. This is a complex issue and individual needs vary from patient to patient. First, triggers that elicit cocaine cravings should be identified and then contingency planning can be arranged. This obviously involves avoiding people and places where there is current drug use, and it is also best to avoid people and places that were associated with previous use. It may involve changing phone numbers or even moving to a new address, to avoid drug dealers who may attempt to entice patients back to cocaine use. It may involve having a friend pick up a pay check and bank the money. It always involves destroying or removing all drug-related paraphernalia and any secret supplies.

Regular attendance at 12-step recovery support groups such as Alcoholics Anonymous (AA), Narcotics Anonymous (NA), or Cocaine Anonymous (CA) is probably the most important aspect of relapse prevention. Other aspects include regular exercise, which helps to normalize brain neurochemistry and combat some of the anhedonia, and stress management techniques to help anticipate stress, recognize it and learn how to feel calm without using drugs.

Involving the family in the recovery process is an important part of treating the abuser. Family members have often been severely affected by the drug use and the behaviors associated with it. Often the families have a lot to learn about drug problems, and experience tells us that if family members are involved with their own recovery process, patients are more likely to recover.

The majority of treatment for cocaine is currently performed in an inpatient setting rather than an outpatient setting. While the hospital setting provides the most security from early relapse, it is not clear if it

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helps with long-term recovery rates. Considerable research is currently underway to attempt to resolve these issues and in the meanwhile it is reasonable to recommend outpatient treatment as an initial treatment if the patient can remain drug-free for a number of days.

Pharmacotherapy

A search has been under way for pharmacotherapeutic aids to help the cocaine addict begin and stay abstinent. A number of drugs which have effects on the dopamine system have been studied.

Bromocryptine (Parlodel®), a drug used mostly in the treatment of patients with Parkinson's disease, is a dopamine agonist. An initial open trial of bromocryptine by Gold and Dackis¹¹ indicated some decreased cocaine craving when bromocryptine was used in a group of cocaine addicts. More recently, however, a double-blind study indicated that almost 60% of the patients taking bromocryptine had to drop out of the study because of side effects and only about 15% continued the study for the required 10 days.¹²

Amantadine (Symmetrel®) has a mechanism of action to increase dopamine's effects, and it, too, is used in Parkinson's disease. It does not appear to have an abuse potential and seems to have a decreased side-effect profile when compared to bromocryptine. Tennant studied 50 patients receiving amantadine in an open trial and reported substantial, immediate reductions in cocaine craving.¹² Two other groups of researchers, however, have reported only a slight decrease in cocaine craving after one to two weeks and these trials had very high dropout rates.¹²

Early animal experiments have indicated that tricyclic antidepressants (TCAs) may be useful in reversing some of the neurotransmitter effects of stimulants. Initially Tennant and Rawson evaluated the effects of desipramine in cocaine addicts, and their initial results looked promising. Since then at least five other open studies have reported generally positive results, with Gawin and Kleber reporting results as high as 80-92% abstinence.¹³ Unfortunately, in all of these studies there has been a delay in onset of effect of 1-3 weeks. A number of double-blind trials have recently been started and results so far have been mixed. In a small study Gawin and others reported initial abstinence rates of 57% with desipramine versus only 17% with placebo,¹³ but other results have not tended to support these findings. It is not clear what effect desipramine may have on long-term abstinence rates. Side effects from TCAs have generally been low, but a recent study of cocaine-induced panic attacks indicated that treatment with TCAs often worsened the problem.¹⁴ In addition, some recent case reports have suggested that TCAs used past 4-5 months after the last cocaine use may produce intolerable anticholinergic effects, dysphoria or even increased cocaine craving.¹⁵

Clearly, an ideal pharmacotherapeutic agent has not

emerged. Ongoing research is continuing with the above agents and others including carbamazepine, clonidine and newer antidepressants.

Conclusion

Cocaine is a very powerful drug and recovery is a difficult and slow process. Current treatment methods are frequently successful and there is reason to hope that in the near future research will enhance these success rates. It is important for physicians to consider cocaine abuse as a problem for any patient they are treating, and to offer them assistance or referral if they need it.

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20-Year Survival of von Hippel-Landau Hemangiomatosis: Case Report

Leo Goldhammer, MD

Arlington, Virginia

VON Hippel-Landau disease is a rare familial disease, transmitted as an autosomal dominant trait, characterized by retinal angiomas (von Hippel tumor), cerebellar hemangioma, and multiple visceral masses, including cavernous angioma of the liver, cyst or adenoma of the pancreas, kidney and epididymis, renal cell carcinoma and pheochromocytoma. We present a patient with von Hippel-Lindau disease who, over a period of 20 years, underwent successful resection of 11 neuraxial hemangioblastomas affecting the frontal lobe, cerebellum, medulla oblongata, spinal cord and retina.

Case Report

About 20 years ago a 43-year-old patient awoke with foggy vision and dark spots in the left eye. Ophthalmologic examination revealed multiple retinal angiomas bilaterally. She underwent retinal surgery but subsequently developed progressive loss of vision leading to complete blindness in the left eye. The following year, neurologic evaluation revealed loss of proprioception and light touch in the left upper extremity. Although the patient underwent posterior fossa exploration, the tumor was considered inoperable and was not resected.

Three years later, the patient complained of paresthesias, weakness and wasting of the extremities, and dysesthesias of the hands and arms. She developed a staggering gait and gradually progressing akinetic mutism.

A ventricular peritoneal shunt was performed five years later. The patient regained consciousness and ocular bobbing, previously noted, stopped the morning after surgery. In three weeks she was able to walk, but demonstrated broad-based staggering and sensory deficit for all modalities in the upper extremities. The optic disk had the appearance of a subsiding papilledema. The patient was alert and well-oriented.

Late in the same year, seven hemangioblastomas

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were removed. These included one large and two small angiomas on the right cerebellar hemisphere, another angioma between the left tonsil and corpus restiformis, two small dorsolateral angiomas with origins at C1 and C2 and one intramedullary angioma (Figs. 1A and 1B, 2A and 2B. The arteriogram did not reveal all of the existing tumors.)

After surgery, there was a continuum of disordered extraocular movements in any direction (opsoclonus). These microsaccades were followed in the ensuing weeks by short bursts of horizontal oscillations (flut-

ter), which were replaced by dysmetria.

Two years later, the patient's condition became slightly worse. One large and two small recurrent hemangioblastomas were successfully removed from the cerebellar hemispheres and anterior vermis. Five years later, a frontal lobe meningioma was resected (Fig. 3).

Family History

The family history confirmed the autosomal dominant inheritance pattern characteristic of von Hippel-



Fig. 1A (left). Film indicates vascular lesion at C2-C3. Feeder arteries (arrows) arise proximally and distally from right



vertebral artery. Fig. 1B. Same view after surgical removal of lesion.

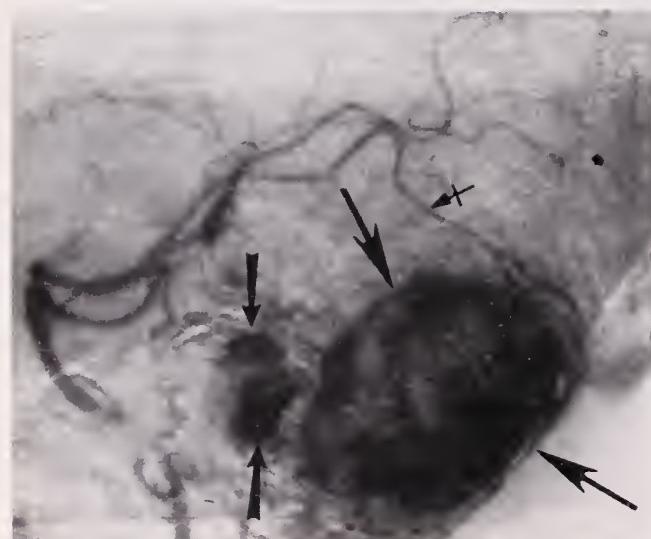


Fig. 2A (left). Preoperative lateral projection of vertebral artery angiogram reveals two distinct vascular mass lesions in posterior fossa (arrows). Larger lesion is supplied by dilated branch of superior cerebellar artery (crossed arrow). Fig. 2B. Postoperative lateral view. Larger hemangioblastoma removed. Second, smaller lesion still visualized (arrows).

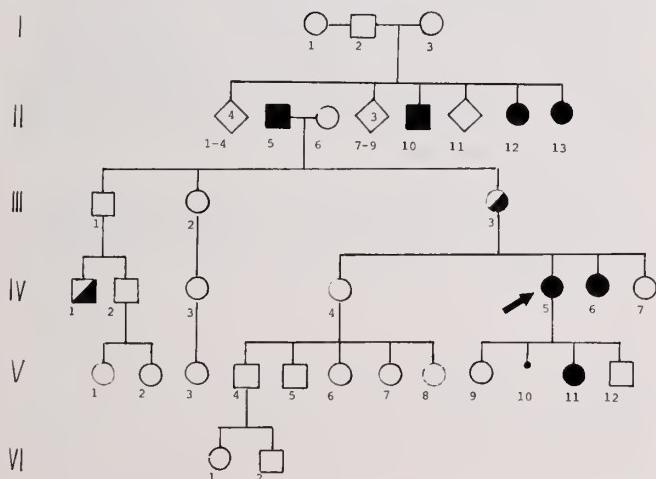




Fig. 3. AP view demonstrates large vascular mass in right frontal lobe.

Lindau disease. In the pedigree (Table 1), the propositus is indicated by the arrow at line IV, number 5. Her sister died at age 24 of pheochromocytoma, preceded by a history of malignant hypertension. Another sister died at 2 months of age of congenital cardiac malformation. The patient's mother died of brain tumor at age 29. The patient's uncle is said to be normal but produced a son who is reported to be blind in one eye, with an angioma as the suspected cause. An aunt at the age of 76 is epileptic but exhibits no other symptoms.

Table 1. Genetic Family Tree of the Propositus.



The maternal grandfather is reported to have died at age 46 of von Hippel-Landau disease. Three of the grandfather's siblings are reported to have gone blind at early ages, presumably due to retinal angioma.

The propositus had four pregnancies and three live-born children. One child, a girl, had a pheochromocytoma and a pancreatic cyst, which were successfully removed. The other two children (V-9 and V-12) are at this time symptom-free.

Comment

Hemangioblastoma is a non-metastasizing tumor which may occur anywhere in the body.¹⁻³ Treatment is entirely neurosurgical, and irradiation does not influence the course of the disease.^{4,5} The best prognosis is associated with angiomatic cysts. Photocoagulation is somewhat effective in the early stages of the disease. A good prognosis is also associated with resection of the hypernephroma before it metastasizes. Onset of symptoms may occur at any time between 3 and 50 years of age and is heralded by either visual or, less often, neurologic disturbances. Few cases have been reported to start in the sixth decade. Patients in older age groups have a more protracted history, sometimes for years, whereas younger patients have a more acute onset of only a few weeks' duration. Sometimes the disease is heralded by mental

Table 2. Screening Program for Patients at Risk for von Hippel-Lindau Syndrome.¹⁴

Family history and pedigree analysis
Physical exam, including blood pressure
Neurological exam
Ophthalmological exam with contact glass
Abdominal ultrasound imaging
Blood cell count
Urine sediment
Urinary catecholamine excretion
Additionally, for exploration of pathological or equivocal findings: cranial and/or abdominal CT scanning or MR imaging, metaiodobenzylguanidine (MIBG) scintigraphy of the adrenal glands.
Annual cranial CT scan for all individuals at risk, first-degree relatives of hemangioblastoma patients, has been recommended. ¹⁵

changes mimicking a frontal lobe tumor. Trauma is implicated in cyst formation and is believed to accelerate the course of the disease. The sequential group of oculomotor disorders (opsoclonus, followed by flutter and finally dysmetria) after removal of the tumors, suggests that these conjugate disturbances of the eyes may be considered as a continuum in which saccadic movements become more and more precise during the recovery process. In spinal cord hemangioblastoma,^{6,11} symptoms are related to location, extent and developmental stage of the tumor. The spinal mass may be asymptomatic before the third decade, after which its usual intramedullary location in the posterior tier is associated with progressive proprioceptive deficits. A subtle distinguishing feature between hemangioblastoma and other intramedullary tumors is the absence of sphincter involvement and sacral sparing in the former. The level of cerebrospinal fluid protein is exceedingly high in spinal cord hemangioblastoma. This is in contrast with levels observed in cerebellar hemangioblastoma in which protein is seldom elevated and then only minimally.⁷⁻¹⁰

Vertebral and aortic angiography utilizing subtraction techniques will enable surgeons to see the anatomic ramifications of the malformation so that arterial feeders can be ligated, sparing the normal circulation. Because vascular malformations are often located on the posterior tier of the cord, thoracic myelography should be carried out in both the supine and the prone positions, otherwise the angioma may be missed.¹¹⁻¹³

Table 2 gives the basic screening program recommended by Neumann et al¹⁴ for patients at risk for von Hippel-Lindau syndrome.

Microsurgical removal is the technique of choice for

operative treatment of central nervous system hemangioblastomas.¹⁴

Conclusion

The case history presented here is notable because of the patient's long-term survival and significant recovery after successful removal of multiple hemangiomas.

The author expresses gratitude for their help to Sondralee Goldhammer and Susan Greenwald.

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Branchial Cysts Presenting as Neck Infections

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and David L. Mobley, MD
Richmond, Virginia

THE branchial apparatus develops during the second week of fetal life and usually has disappeared by the end of the sixth week. Only the first branchial cleft fails to obliterate, portions of it eventually developing into the external auditory canal. First branchial cleft cysts, appearing either in the preauricular area or posterior to the pinna, account for 8% of all branchial cleft cysts.¹ Second branchial cleft defects account for the majority of branchial defects and are found along the anterior border of the sternocleidomastoides. Third branchial cleft anomalies are rare and appear lower in the neck along the anterior border of the sternocleidomastoides and may open into the pyriform sinus. Fourth branchial cleft defects are exceedingly rare.²

The cysts are lined with squamous epithelium which desquamates, collects as debris in the lumen of the cyst, and tends gradually to enlarge the cyst. These same epithelial cells can be identified by fine needle aspiration, aiding the clinician in establishing a diagnosis.^{3,4} The aspirate may appear much like pus, even in noninflamed cysts; the fluid, however, is usually sterile.⁵ Cytological material is usually available for review within 15-20 minutes using the Giemsa stain or several hours using the Papanicolaou stain. Diagnoses to be considered with a lateral cervical mass include metastatic tumors, benign tumors, lymphomas, congenital cysts and abscesses.

Branchial anomalies present at any age and without sex predilection. Typically, patients present in childhood and early adulthood following an upper respiratory infection with a neck mass or cyst, which slowly enlarges. Two percent of the cyst or sinus tracts are bilateral. Inflammation may develop with tenderness or abscess. Sinus drainage cutaneously or into the

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Fig. 1. Epithelial cyst wall with epithelial debris and inflammatory cells in the lumen (photomicrograph X200).

pharynx may occur. Other symptoms include dysphagia and respiratory distress.

The diagnosis of a branchial cleft cyst is straightforward when a child or young adult presents with a cystic mass in the lateral neck area but much more difficult to establish when the initial presentation is an acutely inflamed cervical mass of recent onset. Whether in a child or adult, these cysts are not a common finding. For instance, between January 1968 and September 1989 branchial cleft cysts were diagnosed in only eight patients at the Medical College of Virginia Hospitals. Five of these patients were female and three were male. Six were of adult age. All eight patients had been treated by community physicians for varying lengths of time. All patients had rapid onset of the head and neck mass associated with redness, swelling, and tenderness and were presumed to have abscesses. Three patients underwent incision and drainage, and in one patient the procedure was repeated. Six patients underwent fine needle aspiration. Of note, one patient presented with thrombosis of the carotid artery system secondary to chronic inflammation and infection of the adjacent cyst.

Definitive therapy for branchial anomalies is surgi-

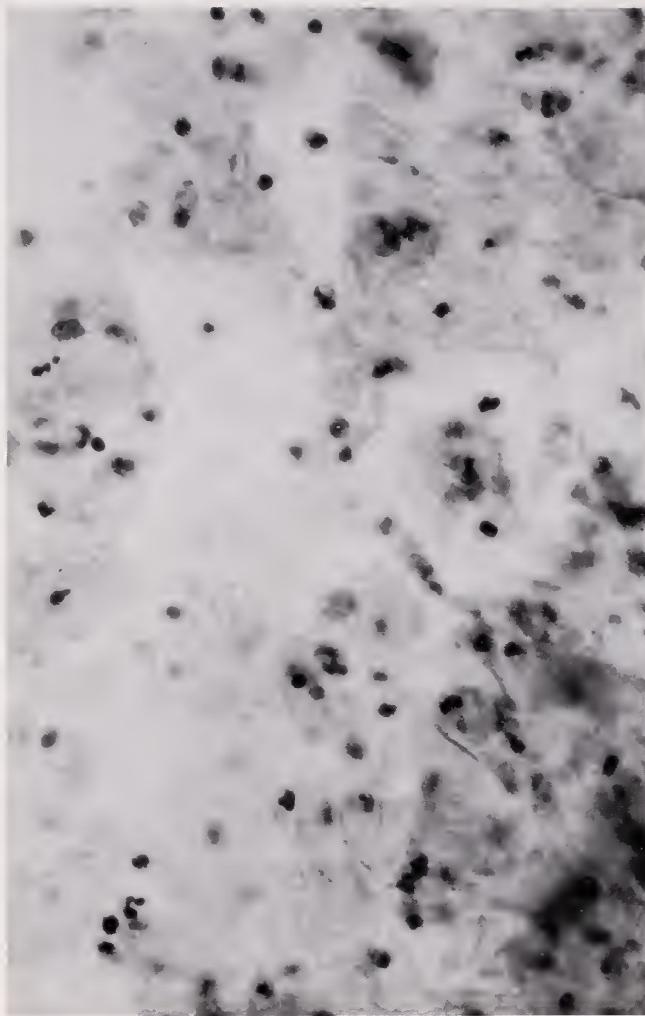


Fig. 2. Squamous epithelial cells benign in appearance (photomicrograph X400).

cal excision. Fine needle aspiration biopsy is often helpful in establishing the diagnosis, particularly if the mode of presentation is unusual. It should be stressed that branchial cleft cysts present not uncommonly as abscesses, and the diagnosis should be considered in any acutely inflamed mass presenting in the head and neck area.

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ABSTRACTS

These abstracts were presented at the annual meeting of the Virginia Neurological Society on February 2 in Williamsburg. Dr. Lawrence H. Phillips II, Charlottesville, was program chairman.

Thrombolytic Therapy for Acute Stroke. E. Clarke Haley, Jr., MD, Charlottesville.

Thrombolytic therapy for acute cerebral infarction has received renewed interest in the latter half of the 1980s. The new, recombinantly-produced thrombolytic agents, such as tissue plasminogen activator (rt-PA), hold the potential for being safer than their previously available predecessors, by virtue of their having enhanced activity in the presence of fibrin clot, thereby avoiding systemic fibrinolysis. Review of recently published series reporting results of intra-arterial thrombolytic therapy with either urokinase or streptokinase suggests that recanalization of acutely occluded cerebral arteries can be achieved in 50% of cases treated in under 24 hours from the onset of symptoms, and that given that rate of recanalization, about 40% of all the patients that were so treated had substantial neurological recovery. The rate of complicating intracerebral hemorrhage was small, but acceptable. Fortified by this experience, four medical centers (University of Cincinnati, Cornell Medical Center, University of Virginia, and Winchester, Virginia, Medical Center) began an NINDS-funded, dose-escalation safety study of intravenous rt-PA administered very early in the course of acute cerebral infarction in 1987. Preliminary observations in the 74 patients accrued to this study will be discussed. Future plans for a randomized trial will also be presented.

The Ticlopidine Story. John W. Harbison, MD, Richmond.

Ticlopidine hydrochloride is a new potent antiplatelet agent that inhibits the exposure of the GP II b/IIIa surface adhesion receptors of platelets by an unknown mechanism. The effect is both dose and time-related with an onset of activity of 24 to 72 hours. Maximal activity occurs at 3 to 5 days, and effect is still noted 72 hours after a final dose. It is well absorbed orally and extensively metabolized with at least one pharmacologically active metabolite. It is excreted in both urine and feces.

Two recent large multi-centered clinical trials have demonstrated significant success in the reduction of stroke risk. In the Ticlopidine Aspirin Stroke Study (TASS), 3069 patients at 56 North American centers with amaurosis fugax, transient ischemic attacks and minor stroke were randomly assigned to either aspirin 650 mg twice daily or ticlopidine 250 mg twice daily in

a double-blind fashion and followed for 2 to 6 years. Using an intent-to-treat primary analysis, the rates of fatal and non-fatal stroke at 3 years were 10% for ticlopidine and 13% for aspirin, a 21% risk reduction ($P = 0.024$). Patients with recent thromboembolic stroke were studied in a randomized, double-blind, placebo-controlled trial of ticlopidine assessing the rate of reduction of recurrent stroke, myocardial infarction or vascular death. The Canadian-American Ticlopidine Study (CATS) entered 1072 patients at 25 North American centers and followed them for up to 3 years. With primary analysis as efficacy, the event rate per year for stroke, MI or vascular death considered together was 15.3% in the placebo group and 10.8% in the ticlopidine group, a risk reduction of 30.2% ($P = 0.006$). The intent-to-treat analysis revealed a risk reduction of 23.3% ($P = 0.020$). Ticlopidine was beneficial in both men and women in both studies. Adverse drug reactions included neutropenia (severe in about 1% of cases), skin rash and diarrhea (severe in 2% of cases each). All were reversible. Both studies revealed benefit from ticlopidine in stroke risk reduction.

Calcium Channel Blockers in the Treatment of Stroke, Subarachnoid Hemorrhage and Migraine. Robert J. DeLorenzo, MD, Richmond.

Calcium is a major second messenger that signals neurotransmitter release and numerous neurochemical processes in the brain, blood vessels and numerous tissues. Because of calcium's universal importance as a second messenger, significant research has been directed at understanding how calcium enters cells and regulates metabolism. Calcium entry into cells has been implicated in regulating numerous neurological conditions. A current theory of one of the major causes of ischemic brain injury during stroke is the accumulation of excessive calcium within neurons. In subarachnoid hemorrhage, calcium can accumulate around the endothelium and lead to vasospasm. Migraine, another important neurological entity, is believed to be caused by spasms in the cerebral blood supply. Calcium entry into the muscles controlling vascular contraction has been implicated in muscle spasms that occur during migraine. Thus, the use of calcium channel blockers to prevent calcium entry suddenly has emerged on the clinical scene for treating conditions where calcium may be causing pathology. At the present time, protocols are being initiated to study calcium channel blockers for the treatment of stroke, subarachnoid hemorrhage and migraine. Significant clinical data is already available on the benefits on calcium channel blockers for the treatment of subarachnoid hemorrhage and migraine. The role of calcium blockers in stroke is still premature but laboratory research as well as human studies suggest that this may also be of significant benefit in decreasing the

morbidity and mortality associated with stroke.

The purpose of this lecture is to provide information about the types of calcium channels and how their regulation might have implications to clinical disease. The material provides a synopsis of what is currently known about calcium channels and calcium metabolism once calcium enters the cell. The relationship of the calcium system to the treatment of stroke, subarachnoid hemorrhage and migraine will be presented in significant detail. It is hoped that this information will be helpful to the clinician in evaluating future research and treatment protocols that will be coming out in the literature utilizing calcium channel blockers or calcium metabolism inhibitors in treating neurological disease.

New Approaches for Management of Stroke. N.F. Kassell, MD, Charlottesville.

Recently, there have been remarkable advances in the management of patients with intracranial aneurysms and arteriovenous malformations and occlusive cerebrovascular disease. The diagnosis of aneurysms will soon be facilitated by perfection of magnetic resonance angiography. Unruptured aneurysms should be corrected if they are larger than 5–7 mm in diameter. Rebleeding and vasospasm are the leading causes of death and disability after aneurysmal subarachnoid hemorrhage. The peak of rebleeding is within the first 24 hours following the initial hemorrhage and is 75% fatal. With modern neurosurgical and interventional angiographic techniques, most aneurysms can be obliterated, regardless of size or location. Surgery should be performed as soon as possible to decrease the possibilities of rebleeding. Accordingly, early diagnosis and referral of patients with ruptured aneurysms to neurosurgical attention is essential to optimize outcome. Transcranial Doppler is a new adjunct for diagnosing cerebral vasospasm. Vasospasm can be prevented by early surgery with removal of clot from the subarachnoid space at the time of aneurysm clipping. Calcium channel blocking agents (nimodipine and nicardipine) and the 21 amino steroids show promise for managing vasospasm. Spastic arteries can be dilated with transluminal angioplasty. Ischemic deficits from vasospasm can be reversed with induced systemic arterial hypertension and hypervolemia.

The incidence of arteriovenous malformations AVMs has been increasing as a result of CT and MR scanning. Most AVMs can now be safely treated with microsurgery, radiosurgery, embolization, or a combination of these modalities. Surgical removal of arteriovenous malformations, even large lesions in eloquent regions of brain, has been markedly facilitated by the operating microscope which assists in identifying feeding arteries and draining veins and maintaining the plane of cleavage of the malformation from the

Ciguatera Fish Poisoning: An Outbreak Associated With Fish Caught From North Carolina Coastal Waters. Peter D. Morris, MD, Douglas S. Campbell, MD, and John I. Freeman, DVM, Raleigh, North Carolina.

Ten persons who had eaten a seafood meal in North Carolina had gastrointestinal and neurologic symptoms suggestive of ciguatera fish poisoning. In five persons, the neurologic morbidity lasted 30 days or longer. The meal included barracuda, dolphin fish (mahimahi), and yellowfin tuna, all of which were caught in North Carolina coastal waters. Analysis of food-specific attack rates implicated the barracuda as the probable cause of the outbreak. We believe this is the first suspected or confirmed report of ciguatera fish poisoning associated with consumption of fish harvested from mainland US coastal waters outside of Florida. Physicians treating patients with a syndrome resembling ciguatera fish poisoning should inquire about consumption of fish not only from areas where the disease is endemic but also from the southeastern US. *South Med J* 1990;83:379-382

brain, and with new bipolar cautery for coagulating vessels and shrinking the malformation. Stereotactic radiosurgery with the Gamma knife, utilizing a single high dose of precisely focused radiation from 201 intersecting beams of cobalt 60 is an effective means for non-invasively obliterating AVMs less than 30 mm in diameter, with results equivalent to open surgery. AVMs too large for stereotactic radiosurgery can be partially embolized, thus rendering them within the size which can be treated with the Gamma Knife. The feeding arteries and the nidus of AVMs can be obliterated with detachable balloons, particulate emboli, acrylate glues, and microcoils, but embolization has not proven successful in totally obliterating most AVMs. However, it can be used to advantage preoperatively to facilitate resection of AVMs as well as to reduce the size of malformations, allowing them to be treated with the Gamma knife.

Angioplasty of the carotid and vertebral arteries in the neck is in its infancy but may prove to be a viable alternative to surgery for use in those patients in whom endarterectomy is contraindicated for technical reasons or medical risks.

Sporotrichoid Atypical Mycobacteriosis. Kathleen Y. Sawada, MD, Richmond.

A 38-year-old white male asthmatic developed a 1.5-cm well-circumscribed nontender nodule at the base of his thumb. The nodule gradually developed serous drainage which then became purulent. He also

noted the onset of five small erythematous less well-defined nodules extending proximally up his left arm. There were no systemic symptoms. The patient recollects an injury at the primary site when he was cleaning his sailboat a week or two prior to the appearance of the first nodule. Medications included prednisone at a dose of 20 mg a day. A skin biopsy showed findings consistent with atypical mycobacterial infection of the skin, including the presence of scattered acid-fast positive organisms with a beaded appearance. Culture eventually identified *Mycobacterium marinum*. Treatment with rifampin and ethambutol was initiated with early improvement in the nodules.

Mycobacterium marinum is the most common of the mycobacteria to produce primary inoculation skin infections. Single or multiple lesions may be present. There is commonly a history of contact with water such as swimming pools or fish tanks. Medical treatment consists of antituberculous drugs, tetracycline, minocycline, trimethoprim-sulfamethoxazole. For small localized lesions, surgical excision is appropriate. *Presented before the Virginia Dermatological Society on November 12, 1989, in Richmond.*

Cervical Epidural Abscess. Daniel Edney, MD, and W. Michael Scheld, MD, Charlottesville.

Spinal epidural abscesses are uncommon, but cervical epidural abscesses are indeed a rare entity. We present a case which typifies the presentation of an epidural abscess.

A 56-year-old black female with a history of alcohol abuse presented with a 3-day history of right-sided neck and shoulder pain after sustaining a fall. Surgical evaluation was unrevealing with no neck instability noted. A fever of 39°C was documented and she was admitted for further evaluation as she refused lumbar puncture. Within hours of admission, she developed bilateral arm weakness and sustained a respiratory arrest requiring endotracheal intubation. CT-myelogram demonstrated an epidural abscess from C2-C5 with complete block at C3. Surgical drainage revealed a large fluid collection anteriorly with abscess and blood cultures growing *S. aureus*. The source was a sinus tract draining a site of chronic osteomyelitis on the plantar surface of the right great toe.

The mechanism of neurologic impairment is thought to be both mechanical and ischemic secondary to compression and vascular thrombosis. Prognosis for recovery relates directly to time until diagnosis and institution of appropriate therapy (*Neurology* 1989; 37:1747, *N Eng J Med* 1975;293:463, others). *Presented before the Virginia Chapter, American College of Physicians, and the Virginia Society of Internal Medicine on March 24, 1990.*

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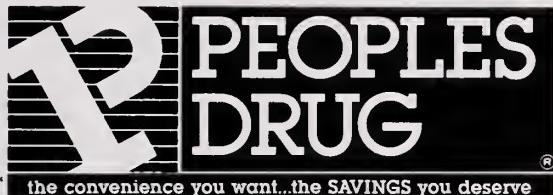
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RICHMOND, VA.

VIRGINIA MEDICAL

Now a Quarterly

BEGINNING with the July issue, VIRGINIA MEDICAL will be published quarterly. At the same time, the Medical Society of Virginia's service-oriented newsletter, now published sporadically, will be increased in size and frequency.

The decision to make this change was made by the Medical Society of Virginia Executive Committee under the leadership of Dr. William H. Barney, President. There was in April unanimous approval by the Medical Society of Virginia Council. There are cogent reasons for the change.

First, present-day print technology permits far swifter dissemination of information than conventional magazine production, which requires a six-week lead time. With the newsletter it will be possible to more promptly disseminate information of news and events of importance to physicians.

Second, scientific manuscripts have dwindled steadily in number during the past decade. No longer are 12 issues a year needed to publish the contributions of Virginia physicians.

The other major factor is cost. The conventional journal format has risen dramatically in cost over the past decade, not only in print and paper, but in postage.

As for the new VIRGINIA MEDICAL QUARTERLY, readers should not anticipate a major change. There will be a new cover, reflecting the change in name. Scientific articles will be continued and manuscripts are solicited. As before, welcome is continued for contributions dealing with the art as well as the economics of medicine. The listing of obituaries and the Who's Who column will continue.

Onward and upward.

E.L.K., JR.

Thanks for a Super Year

THE 1990 General Assembly provided the MSV lobbying team with the most exciting and busiest session I can remember. The legislative team deserves our highest praise and thanks for a super job. Its members dealt directly with over 50 bills, reports, regulations, and the budget. The March 1990 *MSV Legislative Update* listed 26 items, up from 16 for March 1989.

At least ten positive bills were passed, including establishment of peer review standards under the Insurance Commissioner; improved definition of the

Injured Infant Act, as described elsewhere in this issue; exclusion of certain retirement benefits from creditors' claims; broader immunity from liability for HIV infection reporting; nonsmoking areas in indoor public places; additional latitude for the relief of intractable pain. Three bills constructively attacked the combined problems of the un- and under-insured, child health, indigent care and rural health.

This pro-active legislation was all initiated by, contributed to, and/or supported by The Medical Society of Virginia. The Legislative Committee met five times

during the session, and some of its members testified before committees. MSV members owe a great debt to Chairman Ken Tuck and those who donated their time to testify, contact legislators and make telephone

calls. Their efforts were a much needed part of a significantly successful year.

H. C. ALEXANDER III, MD

Collision Course

THE PRIVATE PRACTICE of medicine is on a collision course with the liberals in Congress who espouse an egalitarian approach to the delivery of medical care. They do so with the populist appeal that health care in the United States is at a low watermark. Their solution is to bring about some form of national health insurance and with it federal controls totally ignoring the importance of the free market and the value of competition.

Since the 1930s, beginning with the infamous Wagner-Murray-Dingell bills, American medicine has been continually under siege by liberals in Congress and in academia seeking the lowest common denominator by which medical care will be delivered to the American public. Never mind that the zeal for total equality will ultimately lead to the destruction of the world's best system of medical care; all that is needed, so they say, is to provide for the estimated 37 million Americans without some form of insurance. Their myopia is blinding.

Unless and until individual physicians, irrespective of speciality, unite to protest the egregious addition to title XVIII of the Social Security Act, the fee-for-service practice of medicine will continue on the slippery slope to oblivion. Where better for zealots to begin than with Medicare? This they did by slipping the new section entitled "Payment for Physicians' Services" into the Omnibus Budget Reconciliation Act of 1989 (PL 101-239, Dec. 19, 1989).

This new section provides the Secretary of Health and Human Services with extraordinary powers over the practice of medicine as it applies to the care of Medicare-eligible individuals. The implementation date is 1992. There remains time to influence individual senators and congressmen that the relative value approach to establishing fees is not the American way. Comparable worth as a principle has no place in the free enterprise system of American medicine. What could be worse?

Craftily contained in this section of PL101-239, which did not enjoy wide publicity or extensive testimony, is a not-so-veiled threat of Volume Performance Standards. This has the eerie sound of rationing somewhere after 1992! This, coupled with the other damaging parts of the law, is serious business. How can any physician remain silent when medicine is under such a frontal attack? And what about those who depend on Medicare?

In November 1989 Congress had to admit defeat; it repealed the Medicare Catastrophic Health Care Act passed the previous year. Because of grass roots efforts, senior citizens rebelled and their voices were heard in Congress. Once seniors become aware of the total impact of the 1989 revision to Title XVIII they will rise again in protest that they could well be victims of a medical/surgical rationing plan. An effort is now underway to let senior citizens know that, once again, their senators and representatives have made them targets and hostages to a deliberate effort by liberals in Congress to harness the medical profession and bend the profession to their liberal will.

The Department of Health and Human Services and its Health Care Financing Administration in a report to Congress titled "Medicare Physician Payment" stated (item 6 of the conclusions): "Nonetheless, because the changes in payment that would result under the fee schedule are far more extensive than previous changes, there simply is no reliable basis for predicting the response of physicians either in terms of willingness to treat Medicare patients or willingness to accept assignment. This uncertainty argues for a cautious approach toward fee schedule implementation."

Item 8 of the report's conclusions states: "Although initial results are encouraging, a resource-based fee schedule is not yet ready for use as the exclusive basis for determining all Medicare payments."

The report to Congress from HHS and HCFA is a telling indictment that implementation is fraught with difficulties. This says there is time for grass root protests by physicians and by their Medicare-eligible patients. Without such protests all physicians and all elderly patients will be the losers. With that outcome all Americans will wake up to realize that the health care they have relied on will evaporate into the system "enjoyed" by socialist societies with long waits for care and rationing of needed procedures.

The remark made by the Medical Society of Virginia's President, William H. Barney, MD, in his inaugural address was never more apt: "I am convinced that we are closer now to national health insurance than we have been in the 45 years that I have been in medicine. . ." He was and is so right. Time is short and action is an imperative. Now.

BEDFORD H. BERREY, MD

4431 Old Fox Trail
Midlothian VA 23112

VIRGINIA MEDICAL OBITUARY

Memoir of Levi Hulley 1914-1989

By Thomas W. Murrell, Jr., MD,
Clarry C. Trice, MD, and William R. Hill, MD

Levi Woodbry Hulley, Jr., MD, died in Richmond on December 10, 1989.

Dr. Hulley's early life was spent on Staten Island, New York. He matriculated at the University of Virginia, where he received his BS degree and graduated from the School of Medicine in 1939. After graduation he served internships at the Jersey City Medical Center and the Margaret Hague Maternity Hospital. Much later, in the 1960s, he took courses in aviation medicine at the College of Physicians and Surgeons (Columbia) and Georgetown University School of Medicine.

After serving six months at the Radford Ordnance Works in 1941 he entered general practice in Laurel, Maryland, for one year and then continued practice at Palmyra, Virginia, until 1950, when he moved to Richmond to practice general and industrial medicine. His principle hospital affiliation was St. Elizabeth's Hospital Corporation, where he served as president, chief of staff and director between 1965-1972.

He was a medical advisor to the Industrial Commission of Virginia 1954-1969 and a medical examiner for the Federal Aviation Administration from 1960-1972. Dr. Hulley was also a member of the staff of the A.D. Williams Memorial Clinic at the Medical College of Virginia from 1950-1959.

When in private practice Dr. Hulley was very interested and involved in the Virginia Academy of Family Physicians, beginning in 1951. He served as director, president, treasurer and was a member of a number of its committees. He was also a member of the American Academy of Family Physicians and the Richmond Academy of General Practice.

His involvement in industrial medical practice led him to membership in the Virginia Industrial Medical Society (director, secretary-treasurer 1967-1969), the American Occupational Medical Association, and the Southern Association of Workmen's Compensation Administrators. He was a member of the Richmond Academy of Medicine and The Medical Society of Virginia, serving on multiple committees of both of these organizations, and of the American Medical Association.

In September 1969 Dr. Hulley became medical di-

rector of Blue Cross-Blue Shield of Virginia. Because Levi had been engaged in the practice of clinical medicine this appointment was an excellent choice. During his tenure he exerted maximum effort to be absolutely fair with all disputed claims which were brought to his attention, whether from subscribers or physicians. As an executive of the company he took strong positions against any proposal from without or within which would result in unfair control of the practice of medicine. A number of unfortunate changes in the medical environment occurred after he retired, but he knew he had done his best to stem the tide.

Dr. Hulley was a communicant of All Saints Episcopal Church.

He is survived by his wife, Frances Digges Hulley, one daughter, Dr. Joan Frances Hulley Liverman, and two grandchildren, Eric Louis Hulley Liverman and Astrid Marguerite Bybee Liverman.

Levi Hulley was a man of high ethical standards and a gentleman who will long be remembered in Virginia.

• **John Elwood Collier, MD**, Richmond area physician for 53 years; Medical College of Virginia, 1931; age 83; died April 1, 1990.

• **William Rowland Hill, MD**, Richmond gynecologist and surgeon; University of Virginia School of Medicine, 1934; age 81; died April 10, 1990. Dr. Hill, who retired in January of this year, had been president of The Medical Society of Virginia, the Richmond Academy of Medicine, and the State Board of Health.

• **Lockhart Bemiss McGuire, MD**, Charlottesville, cardiologist and professor of medicine at his alma mater, the University of Virginia (Class of 1957); age 55; died April 29, 1990.

• **Rees Morgan, MD**, captain United States Navy Reserve retired, Ft. Lauderdale, Florida; University of Virginia School of Medicine, 1924; age 89; died February 17, 1990. Before retiring to Florida in 1958, Dr. Morgan practiced obstetrics/gynecology in Roanoke.

• **Eugene Bowie Shepherd, MD**, retired Richmond internist; Medical College of Virginia, 1935; age 82; died April 2, 1990.



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Description: Yohimbine is a 3a-15a-20B-17a-hydroxy Yohimbine-16a-carboxylic acid methyl ester. The alkaloid is found in Rubaceae and related trees. Also in Rauwolfia Serpentina (L) Benth. Yohimbine is an indolalkylamine alkaloid with chemical similarity to reserpine. It is a crystalline powder, odorless. Each compressed tablet contains (1/12 gr.) 5.4 mg of Yohimbine Hydrochloride.

Action: Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalamic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

Indications: Yocon® is indicated as a sympatholytic and mydriatic. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.^{1,2} Also dizziness, headache, skin flushing reported when used orally.^{1,3}

Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.^{1,3,4} 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to 1/2 tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.³

How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

References:

1. A. Morales et al., New England Journal of Medicine: 1221, November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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POSITIONS AVAILABLE in Virginia Beach and surrounding cities—Sentara Medical Care Centers, a subsidiary of Sentara Health Systems, Virginia's premier health care network, are seeking BC/BE physicians to staff its Centers. Family practice opportunities also available. Flexible hours, competitive compensation package, incentive programs available. Excellent recreational, cultural, and educational area. Send CV to Gail Billups, Sentara Medical Care Centers, 22 Koger Executive Center, Suite 202, Norfolk, Virginia 23502 or call collect 804-466-9187. An Equal Opportunity Employer.

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PHYSICIANS WANTED—Family physician or internist for office and hospital practice in the greater Richmond area. There is also an opening for a semi-retired physician for part-time work. Inquiries held strictly confidential. Send CV and financial requirements to Va Med Box 131, 4205 Dover Road, Richmond VA 23221.

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WHO'S WHO

New dean of Eastern Virginia Medical School is Dr. James E. Etheridge, Jr., who had been serving as interim dean since July 1 of last year and before that had been professor and chairman of the department of neurology, specializing in child neurology. His appointment as the school's fifth dean was announced by Edward E. Brickell, EdD, president of the Medical College of Hampton Roads, the administrative body for EVMS.

A native of Norfolk, Dr. Etheridge received both his bachelor's degree in English (1951) and his medical degree (1955) from the University of Virginia, where he also trained in pediatrics. He undertook advanced training in pediatric neurology and neuropathology at the National Institutes of Health, the Mayo Clinic, and Northwestern University School of Medicine and completed two fellowships in clinical electroencephalography, one at the University of Wisconsin School of Medicine, the other at Mayo's. He joined the EVMS faculty in 1974 after teaching assignments at his alma mater and at the University of North Carolina, Chapel Hill.

As dean, Dr. Etheridge will also be provost of the Medical College of Hampton Roads, the next most senior post to the presidency.

The elections of these twelve new component society presidents have been reported to VIRGINIA MEDICAL since the first of the year:

Albemarle County, Dr. Howard A. Montgomery, Charlottesville.

Alexandria, Dr. Frederick M. Stier, Alexandria.

Arlington County, Dr. Philip M. Borges, Falls Church.

Danville-Pittsylvania, Dr. Victor Oberheu, Danville.

Fairfax County, Dr. Brantley P. Vitek, Annandale.

Loudoun County, Dr. Thomas J. Gates, Leesburg.

Mid-Tidewater, Dr. Frederick S. Arnold, Gloucester.

Newport News, Dr. Helmuth W. Triessmann, Jr., Newport News.

Northern Virginia, Dr. Marc E. Read, Woodstock.

Prince William County, Dr. Jared E. Florance, Manassas.

Roanoke Valley, Dr. Samuel J. Williams II, Roanoke.

Southside Virginia, Dr. Joseph L. Crosier, Petersburg.

Tri-County, Dr. Carlos Pinto, Suffolk.

For his "outstanding contribution to the Medical College of Virginia Alumni Association," Dr. Alton R. Sharp, Jr., Richmond, was selected

by the Association's Medical Division for its 1990 Caravati Award. The presentation was made at a dinner and reunion in Richmond.

Dr. William C. Andrews, Jr., Lynchburg, has been inducted as a fellow of the American Academy of Orthopaedic Surgeons.

Fellowship in the American College of Surgeons has been conferred on **Dr. G. Andrew Gehrken, Jr.,** Martinsville.

Dr. Steven M. Zimmer, Arlington, has been installed as president 1989-1991 of the Virginia Thoracic Society.

Dr. Harvey V. Lankford, Richmond, recently received a national recognition certificate from the American Diabetes Association for his outpatient diabetes program, one of only four such programs in the country to receive the designation.

Taylor Dabney



State Visit Virginia's new Secretary of Health and Human Resources invited the Medical Society of Virginia's President to drop by Capitol Square in Richmond for a visit, and they are pictured above, Secretary Howard M. Cullum on the left and Dr. William H. Barney on the right, when Dr. Barney was in Richmond for the Teen Health Forum described elsewhere in this issue. Their conversation was fruitful, and Dr. Barney returned the Secretary's hospitality by inviting him to meet with MSV officers, councilors and selected committee chairmen to discuss urgent medical matters in Virginia.



VASOTEC®

(ENALAPRIL MALEATE | MSD)

VASOTEC is available in 2.5-mg, 5-mg, 10-mg, and 20-mg tablet strengths.

Contraindications: VASOTEC® (Enalapril Maleate, MSD) is contraindicated in patients who are hypersensitive to this product and in patients with a history of angioedema related to previous treatment with an ACE inhibitor.

Warnings: Angioedema. Angioedema of the face, extremities, lips, tongue, glottis, and/or larynx has been reported in patients treated with ACE inhibitors, including VASOTEC. In such cases, VASOTEC should be promptly discontinued and the patient carefully observed until the swelling disappears. In instances where swelling has been confined to the face and lips, the condition has generally resolved without treatment, although antihistamines have been useful in relieving symptoms. Angioedema associated with laryngeal edema may be fatal. **Where there is involvement of the tongue, glottis, or larynx likely to cause airway obstruction, appropriate therapy, e.g., subcutaneous epinephrine solution 1:1000 (0.3 mL to 0.5 mL), should be promptly administered.** (See ADVERSE REACTIONS.)

Hypotension: Excessive hypotension is rare in uncomplicated hypertensive patients treated with VASOTEC alone. Patients with heart failure given VASOTEC commonly have some reduction in blood pressure, especially with the first dose, but discontinuation of therapy for continuing symptomatic hypotension usually is not necessary when dosing instructions are followed; caution should be observed when initiating therapy. (See DOSAGE AND ADMINISTRATION.) Patients at risk for excessive hypotension, sometimes associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death, include those with the following conditions or characteristics: heart failure, hyponatremia, high-dose diuretic therapy, recent intensive diuresis or increase in diuretic dose, renal dialysis, or severe volume and/or salt depletion of any etiology. It may be advisable to eliminate the diuretic (except in patients with heart failure) reduce the diuretic dose, or increase salt intake cautiously before initiating therapy with VASOTEC in patients at risk for excessive hypotension who are able to tolerate such adjustments. (See PRECAUTIONS, Drug Interactions and ADVERSE REACTIONS.) In patients at risk for excessive hypotension, therapy should be started under very close medical supervision and such patients should be followed closely for the first two weeks of treatment and whenever the dose of enalapril and/or diuretic is increased. Similar considerations may apply to patients with ischemic heart disease or cardiovascular disease in whom an excessive fall in blood pressure could result in a myocardial infarction or cerebrovascular accident. If excessive hypotension occurs, the patient should be placed in the supine position and, if necessary, receive an intravenous infusion of normal saline. A transient hypotensive response is not a contraindication to further doses of VASOTEC, which usually can be given without difficulty once the blood pressure has stabilized. If symptomatic hypotension develops, a dose reduction or discontinuation of VASOTEC or concomitant diuretic may be necessary.

Neutropenia/Agranulocytosis: Another ACE inhibitor, captopril, has been shown to cause agranulocytosis and bone marrow depression, rarely in uncomplicated patients but more frequently in patients with renal impairment, especially if they also have a collagen vascular disease. Available data from clinical trials of enalapril are insufficient to show that enalapril does not cause agranulocytosis at similar rates. Foreign marketing experience has revealed several cases of neutropenia or agranulocytosis in which a causal relationship to enalapril cannot be excluded. Periodic monitoring of white blood cell count in patients with collagen vascular disease and renal disease should be considered.

Precautions: General: Impaired Renal Function. As a consequence of inhibiting the renin-angiotensin-aldosterone system, changes in renal function may be anticipated in susceptible individuals. In patients with severe heart failure whose renal function may depend on the activity of the renin-angiotensin-aldosterone system, treatment with ACE inhibitors, including VASOTEC, may be associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death.

In clinical studies in hypertensive patients with unilateral or bilateral renal artery stenosis, increases in blood urea nitrogen and serum creatinine were observed in 20% of patients. These increases were almost always reversible upon discontinuation of enalapril and/or diuretic therapy. In such patients, renal function should be monitored during the first few weeks of therapy.

Some patients with hypertension or heart failure with no apparent preexisting renal vascular disease have developed increases in blood urea and serum creatinine, usually minor and transient, especially when VASOTEC has been given concomitantly with a diuretic. This is more likely to occur in patients with preexisting renal impairment. Dosage reduction and/or discontinuation of the diuretic and/or VASOTEC may be required.

Evaluation of patients with hypertension or heart failure should always include assessment of renal function. (See DOSAGE AND ADMINISTRATION.)

Hyperkalemia: Elevated serum potassium ($>5.7 \text{ mEq/L}$) was observed in approximately 1% of hypertensive patients in clinical trials. In most cases these were isolated values which resolved despite continued therapy. Hyperkalemia was a cause of discontinuation of therapy in 0.28% of hypertensive patients. In clinical trials in heart failure, hyperkalemia was observed in 3.8% of patients, but was not a cause for discontinuation.

Risk factors for the development of hyperkalemia include renal insufficiency, diabetes mellitus, and the concomitant use of potassium-sparing diuretics, potassium supplements, and/or potassium-containing salt substitutes, which should be used cautiously, if at all, with VASOTEC. (See Drug Interactions.)

Surgery/Anesthesia: In patients undergoing major surgery or during anesthesia with agents that produce hypotension, enalapril may block angiotensin II formation secondary to compensatory renin release. If hypotension occurs and is considered to be due to this mechanism, it can be corrected by volume expansion.

Information for Patients:

Angioedema: Angioedema, including laryngeal edema, may occur especially following the first dose of enalapril. Patients should be so advised and told to report immediately any signs or symptoms suggesting angioedema (swelling of face, extremities, eyes, lips, tongue, difficulty in swallowing or breathing) and to take no more drug until they have consulted with the prescribing physician.

Hypotension: Patients should be cautioned to report lightheadedness, especially during the first few days of therapy. If actual syncope occurs, the patients should be told to discontinue the drug until they have consulted with the prescribing physician.

All patients should be cautioned that excessive perspiration and dehydration may lead to an excessive fall in blood pressure because of reduction in fluid volume. Other causes of volume depletion such as vomiting or diarrhea may also lead to a fall in blood pressure; patients should be advised to consult with the physician.

Hyperkalemia: Patients should be told not to use salt substitutes containing potassium without consulting their physician.

Neutropenia: Patients should be told to report promptly any indication of infection (e.g., sore throat, fever) which may be a sign of neutropenia.

NOTE: As with many other drugs, certain advice to patients being treated with enalapril is warranted. This information is intended to aid in the safe and effective use of this medication. It is not a disclosure of all possible adverse or intended effects.

Drug Interactions:

Hypotension: Patients on Diuretic Therapy: Patients on diuretics and especially those in whom diuretic therapy was recently instituted may occasionally experience an excessive reduction of blood pressure after initiation of therapy with enalapril. The possibility of hypotensive effects with enalapril can be minimized by either discontinuing the diuretic or increasing the salt intake prior to initiation of treatment with enalapril. If it is necessary to continue the diuretic, provide close medical supervision after the initial dose for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and DOSAGE AND ADMINISTRATION.)

Agents Causing Renin Release: The anti-hypertensive effect of VASOTEC is augmented by antihypertensive agents that cause renin release (e.g., diuretics).

Other Cardiovascular Agents: VASOTEC has been used concomitantly with beta-adrenergic-blocking agents, methyldopa, nitrates, calcium-channel blockers, hydralazine, prazosin, and digoxin without evidence of clinically significant adverse interactions.

Agents Increasing Serum Potassium: VASOTEC attenuates potassium loss caused by thiazide-type diuretics. Potassium-sparing diuretics (e.g., spironolactone, triamterene, or amiloride), potassium supplements, or potassium-containing salt substitutes may lead to significant increases in serum potassium. Therefore, if concomitant use of these agents is indicated because of demonstrated hypokalemia, they should be used with caution and with frequent monitoring of serum potassium. Potassium-sparing agents should generally not be used in patients with heart failure receiving VASOTEC.

Lithium: Lithium toxicity has been reported in patients receiving lithium concomitantly with drugs which cause elimination of sodium, including ACE inhibitors. A few cases of lithium toxicity have been reported in patients receiving concomitant VASOTEC and lithium and were reversible upon discontinuation of both drugs. It is recommended that serum lithium levels be monitored frequently if enalapril is administered concomitantly with lithium.

Pregnancy - Category C: There was no fetotoxicity or teratogenicity in rats treated with up to 200 mg/kg/day of enalapril (33 times the maximum human dose). Fetotoxicity, expressed as a decrease in average fetal weight, occurred in rats given 1200 mg/kg/day of enalapril but did not occur when these animals were supplemented with saline. Enalapril was not teratogenic in rabbits. However, maternal and fetal toxicity occurred in some rabbits at doses of 1 mg/kg/day or more. Saine supplementation prevented the maternal and fetal toxicity seen at doses of 3 and 10 mg/kg/day, but not at 30 mg/kg/day (30 times the maximum human dose).

Radioactivity was found to cross the placenta following administration of labeled enalapril to pregnant hamsters. There are no adequate and well-controlled studies of enalapril in pregnant women. However, data are available that show enalapril crosses the human placenta. Because the risk of fetal toxicity with the use of ACE inhibitors has not

been clearly defined, VASOTEC® (Enalapril Maleate, MSD) should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Postmarketing experience with all ACE inhibitors thus far suggests the following with regard to pregnancy outcome. Unintended exposure limited to the first trimester of pregnancy has not been reported to affect fetal outcome adversely. Fetal exposure during the second and third trimesters of pregnancy has been associated with fetal and neonatal morbidity and mortality.

When ACE inhibitors are used during the later stages of pregnancy, there have been reports of hypotension and decreased renal perfusion in the newborn. Oligohydramnios in the mother has also been reported, presumably representing decreased renal function in the fetus. Infants exposed *in utero* to ACE inhibitors should be closely observed for hypotension, oliguria, and hyperkalemia. If oliguria occurs, attention should be directed toward support of blood pressure and renal perfusion with the administration of fluids and pressors as appropriate. Problems associated with prematurity such as patent ductus arteriosus have occurred in association with maternal use of ACE inhibitors, but it is not clear whether they are related to ACE inhibition, maternal hypertension, or the underlying prematurity.

Nursing Mothers: Milk in lactating rats contains radioactivity following administration of ^{14}C enalapril maleate. It is not known whether this drug is secreted in human milk. Because many drugs are secreted in human milk, caution should be exercised when VASOTEC is given to a nursing mother.

Pediatric Use: Safety and effectiveness in children have not been established.

REACTIONS: VASOTEC has been evaluated for safety in more than 10,000 patients, including over 2000 patients treated for one year or more. VASOTEC has been found to be generally well tolerated in controlled trials involving 2987 patients.

HYPERTENSION: The most frequent clinical adverse experiences in controlled clinical trials were dizziness (5%), dizziness (4.3%), and fatigue (3%).

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in controlled clinical trials were diarrhea (1.4%), nausea (1.4%), rash (1.3%), orthostatic effects (1.2%), and asthenia (1%).

HEART FAILURE: The most frequent clinical adverse experiences in both controlled and uncontrolled trials were dizziness (7.8%), hypotension (6.7%), orthostatic effects (2.2%), syncope (2.2%), cough (2.2%), chest pain (2.1%), and diarrhea (2%).

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in both controlled and uncontrolled clinical trials were fatigue (1.8%), headache (1.8%), abdominal pain (1.6%), asthenia (1.6%), orthostatic hypotension (1.6%), vertigo (1.6%), angina pectoris (1.5%), nausea (1.3%), vomiting (1.3%), bronchitis (1.3%), dyspepsia (1.3%), urinary tract infection (1.3%), rash (1.3%), and myocardial infarction (1.2%).

Other serious clinical adverse experiences occurring since the drug was marketed or adverse experiences occurring in 0.5% to 1% of patients with hypertension or heart failure in clinical trials in order of decreasing severity within each category.

Cardiovascular: Cardiac arrest, myocardial infarction or cerebrovascular accident, possibly secondary to excessive hypotension in high-risk patients (see WARNINGS, Hypotension); pulmonary embolism and infarction, pulmonary edema, rhythm disturbances, atrial fibrillation, palpitation.

Digestive: Ileus, pancreatitis, hepatitis (hepatocellular or cholestatic jaundice), melena, anorexia, dyspepsia, constipation, glossitis, stomatitis, dry mouth.

Musculoskeletal: Muscle cramps.

Nervous/Psych: Depression, confusion, ataxia, somnolence, insomnia, nervousness, paresthesia.

Urogenital: Renal failure, oliguria, urinary tract infection (see PRECAUTIONS and DOSAGE AND ADMINISTRATION).

Respiratory: Bronchospasm, rhinorrhea, sore throat and hoarseness, asthma, upper respiratory infection.

Skin: Exfoliative dermatitis, toxic epidermal necrolysis, Stevens-Johnson syndrome, herpes zoster, erythema multiforme, urticaria, pruritis, alopecia, flushing, hyperhidrosis.

Special Senses: Blurred vision, taste alteration, anosmia, tinnitus, conjunctivitis, dry eyes, tearing.

A symptom complex has been reported which may include a positive ANA, an elevated erythrocyte sedimentation rate, arthralgias/arthritis, myalgias, fever, serositis, vasculitis, leukocytosis, eosinophilia, photosensitivity, rash, and other dermatologic manifestations.

Angioedema: Angioedema has been reported in patients receiving VASOTEC (0.2%). Angioedema associated with laryngeal edema may be fatal. If angioedema of the face, extremities, lips, tongue, glottis, and/or larynx occurs, treatment with VASOTEC should be discontinued and appropriate therapy instituted immediately. (See WARNINGS.)

Hypotension: In the hypertensive patients, hypotension occurred in 0.9% and syncope occurred in 0.5% of patients following the initial dose or during extended therapy. Hypotension or syncope was a cause for discontinuation of therapy in 0.1% of hypertensive patients. In heart failure patients, hypotension occurred in 6.7% and syncope occurred in 2.2% of patients. Hypotension or syncope was a cause for discontinuation of therapy in 1.9% of patients with heart failure. (See WARNINGS.)

Clinical Laboratory Test Findings

Serum Electrolytes: Hyperkalemia (see PRECAUTIONS), hyponatremia

Creatinine, Blood Urea Nitrogen: In controlled clinical trials, minor increases in blood urea nitrogen and serum creatinine, reversible upon discontinuation of therapy, were observed in about 0.2% of patients with essential hypertension treated with VASOTEC alone. Increases are more likely to occur in patients receiving concomitant diuretics or in patients with renal artery stenosis. (See PRECAUTIONS.) In patients with heart failure who were also receiving diuretics with or without digitalis, increases in blood urea nitrogen or serum creatinine, usually reversible upon discontinuation of VASOTEC and/or other concomitant diuretic therapy, were observed in about 11% of patients. Increases in blood urea nitrogen or creatinine were a cause for discontinuation in 1.2% of patients.

Hemoglobin and Hematocrit: Small decreases in hemoglobin and hematocrit (mean decreases of approximately 0.3 g% and 1.0 vol %, respectively) occur frequently in either hypertension or heart failure patients treated with VASOTEC but are rarely of clinical importance unless another cause of anemia coexists. In clinical trials, less than 0.1% of patients discontinued therapy due to anemia.

Other (Causal Relationship Unknown): In marketing experience, rare cases of neutropenia, thrombocytopenia, and bone marrow depression have been reported. A few cases of hemolysis have been reported in patients with G6PD deficiency.

Liver Function Tests: Elevations of liver enzymes and/or serum bilirubin have occurred.

Dosage and Administration: **Hypertension:** In patients who are currently being treated with a diuretic, symptomatic hypotension occasionally may occur following the initial dose of VASOTEC. The diuretic should, if possible, be discontinued for two or three days before beginning therapy with VASOTEC to reduce the likelihood of hypotension. (See WARNINGS.) If the patient's blood pressure is not controlled with VASOTEC alone, diuretic therapy may be resumed. If the diuretic cannot be discontinued, an initial dose of 2.5 mg should be used under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and PRECAUTIONS, Drug Interactions.)

The recommended initial dose in patients not on diuretics is 5 mg once a day. Dosage should be adjusted according to blood pressure response. The usual dosage range is 10 to 40 mg per day administered in a single dose or in two divided doses. In some patients treated once daily, the antihypertensive effect may diminish toward the end of the dosing interval. In such patients, an increase in dosage or twice-daily administration should be considered. If blood pressure is not controlled with VASOTEC alone, a diuretic may be added.

Concomitant administration of VASOTEC, with potassium supplements, potassium salt substitutes, or potassium-sparing diuretics may lead to increases of serum potassium (see PRECAUTIONS).

Dosage Adjustment in Hypertensive Patients with Renal Impairment: The usual dose of enalapril is recommended for patients with a creatinine clearance $\leq 30 \text{ mL/min}$ (serum creatinine of up to approximately 3 mg/dL). For patients with creatinine clearance $\leq 30 \text{ mL/min}$ (serum creatinine $\geq 3 \text{ mg/dL}$), the first dose is 2.5 mg once daily. The dosage may be titrated upward until blood pressure is controlled or to a maximum of 40 mg daily.

Heart Failure: VASOTEC is indicated as adjunctive therapy with diuretics and digitalis. The recommended starting dose is 2.5 mg once or twice daily. After the initial dose of VASOTEC, the patient should be observed under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and PRECAUTIONS, Drug Interactions.) If possible, the dose of the diuretic should be reduced, which may diminish the likelihood of hypotension. The appearance of hypotension after the initial dose of VASOTEC does not preclude subsequent careful dose titration with the drug, following effective management of the hypotension. The usual therapeutic dosage range for the treatment of heart failure is 5 to 20 mg daily given in two divided doses. The maximum daily dose is 40 mg. Once-daily dosing has been effective in a controlled study, but nearly all patients in this study were given 40 mg, the maximum recommended daily dose, and there has been much more experience with twice-daily dosing. In addition, in a placebo-controlled study which demonstrated reduced mortality in patients with severe heart failure (NYHA Class IV), patients were treated with 2.5 to 40 mg per day of VASOTEC, almost always administered in two divided doses. (See CLINICAL PHARMACOLOGY, Pharmacodynamics and Clinical Effects.) Dosage may be adjusted depending upon clinical or hemodynamic response. (See WARNINGS.)

Dosage Adjustment in Patients with Heart Failure and Renal Impairment or Hypoalbuminemia: In patients with heart failure who have hyponatremia (serum sodium $< 130 \text{ mEq/L}$) or with serum creatinine $> 1.6 \text{ mg/dL}$, therapy should be initiated at 2.5 mg daily under close medical supervision. (See DOSAGE AND ADMINISTRATION, Heart Failure, WARNINGS, and PRECAUTIONS, Drug Interactions.) The dose may be increased to 2.5 mg b.i.d. Then 5 mg b.i.d. and higher as needed, usually at intervals of four days or more, if at the time of dosage adjustment there is not excessive hypotension or significant deterioration of renal function. The maximum daily dose is 40 mg.

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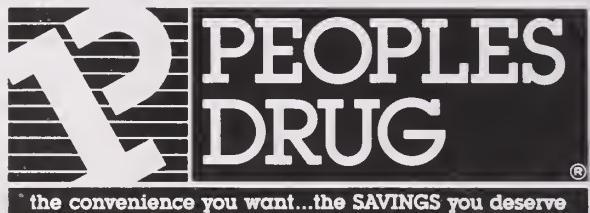
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LETTERS TO THE EDITOR

We welcome your Letters to the Editor.

All letters should be sent to the Editor, VIRGINIA MEDICAL QUARTERLY, 4205 Dover Road, Richmond VA 23221. Please include your name and telephone number. VMQ reserves the right to edit all letters.

Strongly favors privacy for hospital patients/staff

I refer to the Point of View commentary in the April issue in which the author laments the "total demise of hospital wards."¹

Having been a student, intern and resident in the time of open wards (some in poor locations), I would put in a strong statement in favor of privacy for patients and staff in hospitals.

To equate economy-class airline space with wards in hospitals is spurious. Even economy might not apply to both. Certainly economy class by way of wards in hospitals should not be imposed. Furthermore, arriving at the same time on a plane, whatever the class, is a different matter from being treated and trying to recover on a ward.

Some people require privacy. For those who do not need or wish to have solitude, this is a foreign concept, impossible to explain on the one part or to understand on the other.

There remains a place in the world, medical and otherwise, for A Room of One's Own.

Maryjane Luke, MD

Box 351, Glencairn
Covington VA 24426

1. Monteo JM II. Point of View: The case for economy class. Va Med 1990;117:165

Dr. Montero replies: Sadly, the uninsured and underinsured segment of our population no longer has a choice of a less expensive room and, for that matter, ward. Isn't a choice of affordability the essence of our free enterprise system?

Spotlight on cord blood transplant reminds them of a Va Med first

Considerable recent attention has been given to the use of umbilical cord blood for bone marrow transplantation by the medical literature¹ as well as the lay press and television. This letter is written to address the fact that it was the VIRGINIA MEDICAL MONTHLY that published the original article on this usage in 1972² and raised the possibility of umbilical cord blood banks. The clinical work was performed in Petersburg, Virginia, and the laboratory studies in Atlanta, Georgia. The article reported a patient with acute lymphoblastic leukemia who obtained a documented temporary graft following administration of a relatively small quantity of umbilical cord blood matched only for ABO typing.

The use of umbilical cord blood and the potential for cord blood banking has met with considerable resistance. The original publication in 1972 has been largely ignored,^{1,3} and even at Chernobyl it apparently was not considered, fetal liver cells instead being utilized

unsuccessfully when bone marrow was not available.⁴ Following Chernobyl, we attempted to revive the concept that umbilical cord blood holds potentially equal or significantly greater capability for marrow transplantation than does human adult blood marrow, as reported in two more recent articles verifying this basic concept.^{5,6}

Twenty years ago no untoward effects were noted in the 17 terminal patients to whom one of us had given umbilical cord blood late in the progress of their malignancies, including the patient with acute lymphoblastic leukemia mentioned above.⁷

When umbilical cord blood banks and the use of cord blood for marrow transplantation come into existence, the future of medical therapy will be significantly altered, and the article in this journal 18 years ago will, we believe, have led the way.

Norman Ende, MD

New Jersey School of Medicine
Newark NJ 07103

Milton Ende, MD

121 South Market Street
Petersburg VA 23803

1. Gluckman E, Broxmeyer H, Auerback AD et al. Hematopoietic reconstitution in a patient with Fanconi's anemia by means of umbilical cord blood from an HLA-identical sibling. *N Engl J Med* 1989;21:1174-1178
2. Ende M, Ende N. Hematopoietic transplantation by means of fetal (cord) blood—a new method. *Va Med Mon* 1972;99:276-280

3. Broxmeyer HE, Douglas GW, Hangoc G et al. Human umbilical cord blood as a potential source of transplantable hematopoietic stem-progenitor cells. *Proc Natl Acad Sci USA* 1989;86:3828-3832
4. Linnemann RE. Soviet medical response to the Chernobyl nuclear accident. *JAMA* 1987;258:637-643
5. Ende N, Rameshwar P, Ende M. Fetal cord blood's potential for bone marrow transplantation. *Life Sci* 1989; 44:1987-1990
6. Ende N, Giuliani D, Ende M, Ponzio NM. Production of human to mouse xenografts by umbilical cord blood. *Life Sci* 1990;46:1373-1380
7. Ende M. Lymphangiosarcoma: report of a case. *Pac Med Surg* 1966;74:80-82

Cites benefits of prescribing powers for nurse practitioners

What an irony it is that during the same month that VIRGINIA MEDICAL lamented editorially the unavailability of medical care for a significant number of Virginia citizens,¹ The Medical Society of Virginia actively discouraged the bill that would extend prescriptive authority to nurse practitioners, thereby contributing to its carryover.²

There are a significant number of Virginia citizens whom medical doctors cannot and will not serve. Some of these citizens might well be served by nurse practitioners, especially in the area of obstetric and

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gynecological care. One of the barriers nurse practitioners encounter in assuming some of this care is their lack of prescriptive authority.

Instead of opposing the extension of prescriptive authority to nurse practitioners, the Society should take the active step of encouraging it. This step would not hurt us. There are not enough nurse practitioners to have significant negative impact on the practices of most physicians in the state. In fact, supporting extension of prescriptive privileges would result in very favorable publicity. The public would see that we are interested in improving the care of patients and serving their best interests, and not strictly serving our own interests. The favorable publicity from this position might also help us in fighting other more noxious proposals, such as mandatory assignment and the extension of prescriptive authority to optometrists, pharmacists and chiropractors.

The Society has a chance to do something that would confound its critics and not hurt itself and be of great help to the medically underserved citizens of Virginia. It can do this by strongly endorsing and supporting the extension of prescriptive authority to nurse practitioners.

James W. Turner, Jr., MD

2942 Hunter Mill Road, #102B
Oakton VA 22124

1. Morey DAJ. Access: Will we nibble at the edges or bite the bullet? (point of view). *Va Med* 1990; 117:66-67
2. MSV Legislative Update. February 1990, p 2

More waste for the hauler: the ironic polybag

I read with interest your April article on waste disposal¹ while noticing the ironic use of a plastic wrapper for mailing.

One should never criticize without a better solution. Therefore, I refer you to the enclosed brown paper wrapper used to dispatch a recent issue of *The Scientific American*.

Donald S. Daniel, Jr., MD

3540 Floyd Avenue
Richmond VA 23221

1. Keene JH, Gray A. Waste disposal: How to handle the new regulations. *Va Med* 1990;117:144-147

Retirement salute backfires, wounding five

I read in the May issue of VIRGINIA MEDICAL that I have retired!¹ I do wish someone had called me before this was printed in the magazine.

On October 8, 1989, the community of Boykins gave an Appreciation Day on my behalf, which was quite

nice and I enjoyed very much until the local newspaper printed a large headline in error that it was a retirement party. This information was retracted in a later issue but apparently lives on.

I am actively engaged in my practice, working 60 or more hours a week and at the present time have not considered quitting or leaving the community without a physician. I would appreciate an acknowledgement that I am not retired because it has caused great confusion.

J. A. Naranjo, MD

PO Box 365
Boykins VA 23837

1. Who's Who: Retirements. *Va Med* 1990;117:196-199

The Editors greatly regret the published misinformation about Dr. Naranjo.

We apologize also to all concerned for identifying Dr. John P. Heatwole's son as Dr. Kenneth M. (for Martin) Heatwole, the Charlottesville pulmonary specialist, when in truth he is Dr. Kenneth M. (for Michael) Heatwole, the Mechanicsville family practitioner.

And we are distressed to hear from Dr. John M. Stirewalt that the biographical material we published about him was incomplete and partially incorrect. He sends a revised version, herewith printed in full:

A medical career spanning both private and public health ended last year when Dr. John M. Stirewalt of Waynesboro retired from Western State Hospital. He served his entire professional career in Virginia and was at Western State for 22 years.

Born in Harrisonburg, Dr. Stirewalt lived his childhood in New Market, Virginia, and completed high school in Harrisonburg. He attended the University of Virginia undergraduate and medical school (Class of 1951) and completed one year of ENT training at the University's hospital. He first served as staff physician for Woodrow Wilson Rehabilitation Center and later as chief of medical services, then changed to private practice in Waynesboro. Returning to state employment, he served as assistant director for the Augusta/Staunton/Nelson/Highland County Public Health District (1963-1966). At Western State, which he joined in 1966, Dr. Stirewalt filled both clinical and administrative posts. He served as chief of staff in the Medical Center. In 1971, Western State Hospital established a specialized Geriatrics Receiving Unit, which developed into a 500-bed certified psychogeriatric facility of the hospital. In July 1976 he became its director of medical services.

Since 1953 Dr. Stirewalt has been an active member of the Augusta Medical Society and delegate to The Medical Society of Virginia for 12 years. He was honored by election to serve as president of the Augusta/HIGHLAND Medical Society in 1981. As a member of the Western State Hospital medical staff, he was appointed to the teaching faculty of the University of Virginia School of Medicine, Department of Behavioral Medicine and Psychiatry, in 1981 and still serves in this capacity.

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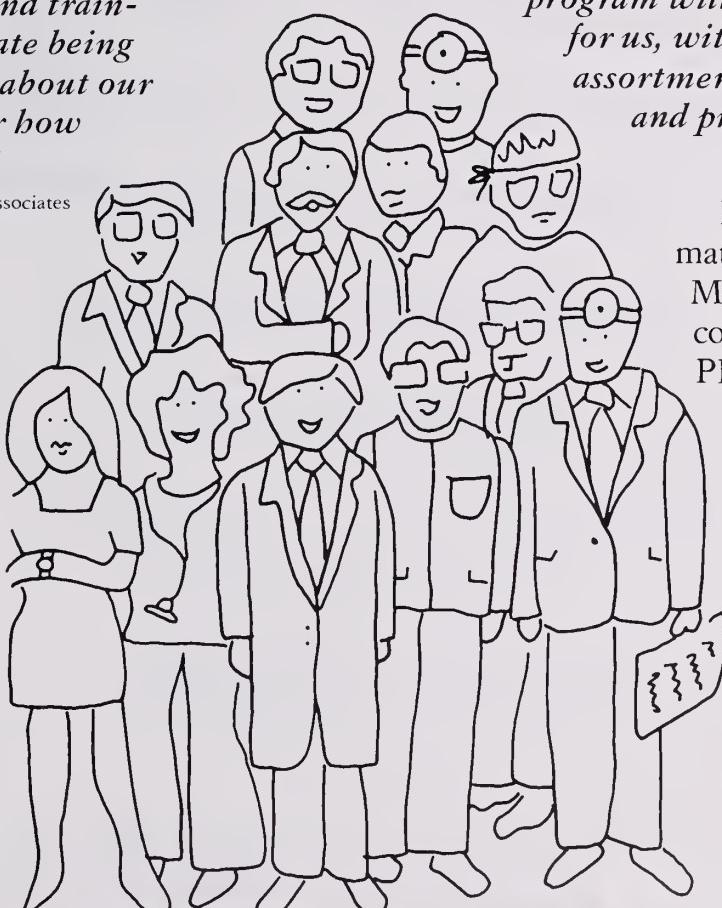
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William H. Barney, MD

New MSV Media Mix

With this issue the journal changes from the VIRGINIA MEDICAL MONTHLY to the VIRGINIA MEDICAL QUARTERLY (VMQ).

The reasons for this change were outlined by the Editor, Dr. Kendig, in his editorial in the June issue.¹ The two primary reasons are the escalating costs of the publication of the journal, together with the declining submission of scientific articles. I know that many of us regret that this change is being made, but the alternative was to discontinue the journal altogether, and I think that this change does provide some positive opportunities for better communications with our members.

The new VIRGINIA MEDICAL QUARTERLY is to be published in January (Winter), April (Spring), July (Summer), and October (Autumn) and will be similar in format to that published on a monthly basis. Some effort will be made to increase the number of features, particularly human interest stories as they involve various groups of physicians.

In each of the eight months that the journal is not published, the Medical Society of Virginia staff

will provide a newsletter, which will be a four- to six-page publication and prepared in-house with minimal publication costs. This will provide a vehicle for officers and committees of the Medical Society of Virginia to communicate with our members on a more timely basis. The lead time required to get an item in the newsletter will be about a week, compared to about six weeks for the printed journal.

In the survey last year inquiring into issues about which MSV members are concerned, it was apparent that there had been a lack of communication between our leadership and our members, and this is an opportunity to correct that deficiency. All members of the Medical Society of Virginia are invited to avail themselves of the journal, as well as the newsletter, to publish timely articles and announcements which may be of interest to our members.

A handwritten signature in cursive ink, appearing to read "William H. Barney".

1. Kendig Eljr. Now a quarterly. Va Med 1990;117:265

The Capitol Connection

JOHN WARNER charged through the doorway and started politicking like a man who is 15 points down in the polls and needs every vote he can get. No matter that he is totally unopposed for reelection, the veteran senator worked the room for all he was worth, clasping hands, clapping backs, laughing, listening, laying on the eye-to-eye contact. It was a virtuoso performance. The doctors loved it, and Warner's peers from the House smiled indulgently.

The setting was the Medical Society of Virginia's annual Congressional Luncheon, with which the Society's leadership honors Virginia's legislators on Capitol Hill, staged this year on May 9 in the Gold Room of the Rayburn House Office Building. Twenty-seven MSV officers, councilors, and AMA delegates made the trip. All eight of Virginia's congressmen were there, sitting at lunch with the councilors from their districts, while Senators Warner and Robb flanked MSV President William H. Barney at the head table.

A week earlier, the Medical Society of Virginia Auxiliary had also connected with some high-ranking Washingtonians. For pictures of that event, turn to page 290.

Clockwise from top left, Senator Warner is shown with Rep. Frederick C. Boucher (seated) and Dr. James L. Patterson, Jr.; Dr. Richard L. Fields (back to camera); Dr. H. Alan Bigley, Jr.; Rep. L. F. Payne, Jr. (center), and Dr. Edwin J. Harvie, Jr.; Dr. William H. Barney (right), Dr. Louis D. Parham (foreground), and aide Rem Dickinson (behind Warner).





Photographs by Mark Charrette

Capitol Connection, Auxiliary Style

THE CELEBRITIES the Medical Society of Virginia Auxiliary fielded during its annual convention were Marilyn Tucker Quayle, wife of the Vice President, and Nancy Sununu, whose husband is White House chief of staff. In a jostle of Secret Service men, the two women arrived to speak at the Auxiliary's post-convention workshop on May 2 at a hotel in Washington's environs. Mrs. Victor N. Guerrero, newly elected MSVA President, ushered them into the room.

Mrs. Quayle dazzled. She has selected breast cancer as the focus of a personal campaign, and she spoke on behalf of education and detection. Mrs. Sununu encouraged, speaking in praise of good works. "Where would we be if all the volunteers in this country disappeared overnight?" she asked. Her audience of 130 volunteers knew the answer to that.

Then the two honored guests departed, and the Auxiliary members settled down to such mundane matters as new techniques for increasing membership and how to run a meeting.



Above, Heidi Guerrero (left) introducing Mrs. Quayle.
In the background, Dr. John A. Owen, Jr., MSV President Elect.
Below, Mrs. Sununu (left) and Mrs. Quayle at the microphone.



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WHO'S WHO

Dr. F. J. Pepper, Alexandria, is the new president of the Virginia Board of Medicine. His election culminates eight years of Board membership, including service as vice president and secretary-treasurer. A native of Omaha, Nebraska, Dr. Pepper was graduated from the University of Nebraska College of Medicine in Omaha (Class of 1965) and performed his residency in psychiatry at St. Elizabeth's Hospital in Washington DC.

Elected vice president was **Dr. Read F. McGehee, Jr.**, Richmond, whose specialty is pulmonary diseases. Tony C. Butera, Alexandria podiatrist, was reelected secretary-treasurer.

Dr. Owen W. Brodie, Jr., walked out of his office at Richmond's Westbrook Hospital late last year, motored to the airport, climbed into his single-engine Cessna Cardinal, and flew off into the blue October sky. Thus ended Dr. Brodie's 21 years of private psychiatric practice at Westbrook, and thus began his career with CompHealth, a national locum tenens firm, and his presidency of

the Flying Physicians Association, both of which have kept him on the wing ever since.

"I've wanted to fly ever since I was a kid in Lynchburg, watching Piedmont's DC3s coming into Preston Glenn Airport," Dr. Brodie remembers, "and if I'd had good enough eyesight, I would have made a career of flying." Instead, he set his sights on medicine, graduating from the Medical College of Virginia in 1962 and taking a residency in psychiatry at Duke University. He established a private psychiatric practice at Westbrook Hospital in 1968, serving successively as director of its geriatric and adult intensive care programs and as president of the medical staff. He has been president also of the Southern Psychiatric Association and the Neuropsychiatric Society of Virginia and has contributed vigorously to an array of national, regional, and local medical societies, including the Medical Society of Virginia and the Richmond Academy of Medicine.

In 1972 Dr. Brodie started taking flying lessons, in 1974 he got his pilot's license, and in 1976 he earned

his instruments rating. Most of the 1,055 members of the Flying Physicians Association which he now heads are licensed pilots, although that is not a requirement of membership. The Association's annual meeting this year is in Vancouver, Canada, and in August Dr. Brodie's Cessna will take him and his wife Pat there. Two other Virginians have been president of the Flying Physicians Association: **Dr. Paul A. Woods**, Waynesboro, and **Dr. Benjamin H. Word**, Charlottesville.

As for the locum tenens practice, "Pat and I love to see new places, meet new people," says Dr. Brodie, and they're having a grand time flying about the 50 states and doing just that.

New president of the Norfolk Academy of Medicine is **Dr. Randolph J. Gould**, Norfolk surgeon.

For his "twin hallmarks of service, medicine and education," **Dr. William F. Bernart**, Nassawadox, was selected by the Eastern Shore Chamber of Commerce for its 1990 Outstanding Citizen of the Year award. Dr. Bernart has practiced on the Eastern Shore for 33 years, and has been active with numerous health fairs, the Northampton-Accomack Memorial Hospital's Auxiliary, and its School of Nursing. The father of five children, he has been a member of the Northampton County School Board, the Eastern Shore College Foundation, and Project Horizons, a group that identifies talented middle school children and seeks to insure that they are offered higher education.

As part of a White House program to encourage volunteerism, President George Bush has honored a Richmond pediatrician who volunteers some 30 hours a week as medical director of the Richmond Cere-

President Brodie and wings.



Photo courtesy *The Flying Physician*

**The Medical Society of Virginia
Annual Meeting '90
The Homestead
October 31–November 4**



bral Palsy Center. He is **Dr. Gayle G. Arnold**, who was selected as a "Point of Light," the 114th chosen since the program was launched. In private practice in Richmond since 1951, Dr. Arnold is president of the American Academy of Cerebral Palsy/Developmental Medicine.

New president of the Psychiatric Society of Virginia is **Dr. John O. Hurt, Jr.**, Salem.

Dr. John M. Daniel III, Richmond, has been elected president of the Virginia Society of Internal Medicine.

Honorary degrees were conferred on four persons at the College of William and Mary's commencement exercises in mid-May, and **Dr. Janet Kimbrough** of Williamsburg was one of them. The 88-year-old retired physician received a doctorate of humane letters. Dr. Kimbrough was the sixth generation of her family—descendants of St. George Tucker, a W&M student in the 1770s and the college's second professor of law in 1790—to attend the college, graduating Phi Beta Kappa in 1921. The other honorees were Virginia's Gov. L. Douglas Wilder, Sir Antony Acland, British ambassador to this country, and D. Tennant Bryan, chairman of Media General.

Last year he won the National Interscholastic Athletic Administrators Association's Distinguished Service Award (Va Med March 1990). This year **Dr. H. Joseph Williams**, Staunton, was named 1990 Rotarian of the Year by Staunton's Rotary Club for being "an outstanding role model for others in the community and in the Rotary Club." Dr. Williams retired recently after 40 years as team physician for the football and basketball teams of Robert E. Lee High School in Staunton.

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3. Health Plan shall have the right to make percentage reductions, and distribution of income for in Attachment A pursuant to Health Plan's present and related rules and regulations.

3.4 If Health Plan becomes insolvent or fails as required by this Agreement for any reason, Physician shall agree that his or her obligations under this Agreement shall continue in full force and effect with respect to Enrollees assigned to him or her for a period of thirty (30) days from the date of Health Plan's insolvency or failure to pay. This provision is not intended, however, to preclude Physician from receiving compensation for services pursuant to insolvency insurance coverage obtained by Health Plan, documents governing state reserves, other health insurance or prepaid policies or other appropriate sources other than Enrollee.

IV. COMPLIANCE WITH "EALTH PLAN RULES

benefit plan described in the sample Evidence of Coverage, shall be paid to Physician in a Physician Manual or separately in the case of application from time to time. In addition, a specific Evidence of Coverage for a particular plan of benefits and copayments shall be paid to Physician upon request.

"Physician Services" means those Health Care Services set forth in evidence of Coverage which might be rendered by a physician.

"Participating Physician" means any physician, pathic physicians, who is duly licensed to Commonwealth of Virginia and who satisfies participation criteria established by the Health Plan and has entered into a contractual agreement with, or is otherwise engaged by Health Plan to provide physician services to Enrollees as either a Primary Care Physician or a specialty physician as defined hereinafter.

A Cautious Inquiry

PHYSICIAN AGREEMENT

PHYSICIAN AGREEMENT
entered into by and between Southern Health Services,
the Commonwealth of Virginia (hereinafter organization to
and the laws of the Commonwealth of Virginia ("Physician duly
sician").
plan is obligated to provide or arrange for the
tain health care services to enrollees in its plan;
enter into an agreement with enrollees for the delivery of health care services to enrollees of Health Services, Inc., and
Managed care plan
but you may or
warns the author, who
before signing

cian upon entering into this Agreement, satisfies
a for Provider participation; and
Plan and Physician mutually desire to preserve and
comises and mutual covenants
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I. DEFINITIONS

I. DEFINITIONS
In this Agreement, the following terms shall have the meanings specified.

"Enrollee" means any person who has been entered into an agreement to such person of medical and hospital service plan.

"Health Care Services" means those benefits to be provided to enrollees under each Enrollee's Evidence of Coverage. The Health Plan has the right to modify such Evidence of Coverage, including the care services described therein, and shall provide notice to plan members of changes to which physician shall be bound. A copy of the Evidence of Coverage, accompanied by a summary of all benefits, and deductible amounts distinguishing certain benefit plans from

300 VIRGINIA MEDICAL QUARTERLY, Summer

XI. ADMINISTRATION

I agree to cooperate and participate with Health Plan in Management Program as described in Attachment E and in prolifee and Provider Grievance Procedure as described in these programs may be altered and supplemented by rules and regulations. Furthermore, Physician shall first determination rendered by such Programs or I agree that Health Plan

that Health Plan may include his or her name, number, type of practice, and facilities as to whom or unwillingness to accept new patients in participation. It is being intended that such roster may be inspected by and is intended to be from time to time by contractor and by others, and other providers, that no

With further documents for this purpose, I expect to collect all information necessary to provide other details of the policy or fund less than the Physician's Plan to fit the bill to the third party all efforts paid.

Care, *The* **Primary** **not** **re-**
Physician **may** **refuse** **to** **see** **the** **Patient**
if **he** **is** **not** **seen** **by** **a** **Physician**

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or him or her in his or her

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with the plan as
Date:

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the services were rendered,

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Health Plan appropriate to
AND FACILITIES
Physician rosters as a Pri-
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comply with all applicable
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EDUCATION

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RECORDS AND FACILITIES

records of Health Plan Enrollees as to comply with all applicable confidentiality of patient records.

THE last 15 years have been a real learning experience. I'm in a ten-person family group practice, all Board certified. Our training and experience is just that—practicing family medicine. That's what we have all wanted to do—and hope to continue. We are old and young, with ages spanning from 30 to 60.

Ten years ago we were a five-person practice, and ran a family practice training program. Then Managed Care came to town. Up to then insurance was pretty simple: file for indemnity insurance and bill the patient what insurance didn't pay. If there was a problem, the patient took it up with his company. Sounds cool. Of course we could add Medicare. Accept it or not, it didn't seem to make much difference, so we accepted it. And add Medicaid: We were already doing our share of free medicine so why not sign up for Medicaid and get paid a little? No problem.

I'd read somewhere that we were in a "targeted" area for managed care plans. Other than being a little curious, I went on to the sports page.

Here they came.

Plan A.

An HMO, owned and run by a big corporation.

"All you have to do is sign up" and "we'll deliver you bunches of patients" and checks every month, guaranteed, based on a "capitation rate."

We met as a group and discussed it. The smarter ones in the group (not me) admitted they didn't really understand all the implications and would rather take a wait-and-see approach. We not-so-smarts prevailed, stating we could learn by being part of it. So we joined.

Plan B.

An HMO owned by physicians.

Even better than Plan A because you'll not only make money practicing medicine, you'll make money on "the plan" by being an owner. Of course you'll have to put up a few

by John A. Mapp, MD

thousand to get it going. And, listen to this, they had better cap rates than Plan A, and higher co-pays to reduce patient abuse. We met as a group and this time even the smarter ones were lured by the thought of being an owner, making money both ways. We joined.

Plan C.

Here's a nifty twist. It's an HMO, but the HMO does its annual cap rate to an IPA. You join the IPA and the IPA then does business with you on a discounted fee-for-service basis.

"Well, what would we be joining?" we asked, "an HMO or a PPO?"

"Well, it's kind of both," and "with the advantages of each," came the reply. We met. We joined.

Plan D.

Forget the description. We met. We joined.

Plan E.

We joined.

Plan F.

Guess what, we didn't join! It might have been a good plan or a terrible one. It didn't really matter because by then we were entirely too confused and tired just trying to sort out the tangle of the ones we were in.

Why?

What brought these on? We were so comfortable before. The answer is both simple and complex. The spiraling cost of medicine is the simple answer, and it's true. Any insurance plan has to keep up with costs by raising the premium. Raising the premium hurts after a while, both the individual and the employer

Address correspondence to Dr. Mapp at First Colonial Family Practice, 1120 First Colonial Road, Suite 100, Virginia Beach VA 23454.

who's paying a part of the premium. Especially if the premiums double two years in a row.

The complex answer lies in the why's of the spiraling cost increases, and I won't get into all of this. The costs of rapid technological changes and improvements; hospitals charging on a cost plus basis; unusual but true cases of physician overcharges; labor costs; medical malpractice premiums. It all adds up to *expensive*. So much so that a large segment of our population doesn't have *any* medical insurance. Too poor to pay the premium and too rich to be eligible for Medicaid.

So what happened?

We learned, bit by bit. Plan A was rolling along. Our committee met with their committee, and we made some adjustments and they made some adjustments.

Read a "hold harmless" clause some time. It should be simple. The "plan" should not be responsible for your goofs and physicians should not be responsible for theirs. That's it. On one occasion we had two lawyers (ours and theirs) arguing over the interpretation of the legal-eze written into the hold-harmless clause. We finally got it simplified.

Then something happened with Plan A. Committees' recommendations started falling on deaf ears, and "corporate decisions" came in the mail. The crowning blow came when our "withhold" percentage went up. It not only went up, it went up *retroactively*. They actually sent us a bill. What a learning process; and in the fine print they had us. Well, we got mad, and we got out. And we wrote our patients and told them why. It hurt for a while, but 95% of those patients are back with us under another plan. Other physicians' practices left Plan A also, and Plan A is not much of a player in our service area any more.

"Withholds" are a particular irritant to us. If the plan makes money we're supposed to get a good percentage of the "surplus" back. It's a financial incentive to prevent us from "over-testing" and "over-re-

ferring." It's also an incentive to "under-test" and "under-refer." We call it a conflict of interests.

Many plans come into an area knowing they're going to lose money overall for three to five years. It's their "loss leader" to get into a particular area, but they still play the withhold game with physicians. We finally came to grips with this by treating withhold as extra discounts and then deciding if we can make a fair profit with that particular plan. That way we practice medicine the way we want and according to what we think is best for the patient.

Plan B. Remember, that's the physician-owned one. It was rolling along okay, too, for a while. Being physician-owned, we had more input and even had a physician director. We made adjustments—lower withhold, better cap rates, differential co-pays according to office hours, after hours "urgent" situations, etc.

Only one problem, Plan B lost money and we owned it. And we (about 150 physicians) didn't have the deep pocket to carry it through. A good-sized corporation eventually bought it and then an even larger one bought it again. It's still around, but not with our physician director or our committee input. I hear it's still losing money.

We did solve a problem regarding co-pays with this plan. Can you imagine the potential for patient abuse of a plan with a \$3 co-pay for anything, regardless of urgency or appointment, or of time of day, night, weekend, or holiday? One patient, I'll call him Bill, used to come in rather regularly on Mondays for his "flu" and to get his work "sick slip." Definitely worth \$3 to have Mondays off. Bill had outsmarted the plan.

Our variable co-pay according to appointment, no appointment, urgency, time of day, weekend, etc., finally straightened this kind of abuse out. Of course, 95% of patients don't abuse plans. They sim-

ply want good medical care without paying a fortune in premiums.

Plan C. Remember the one that's kind of an HMO and kind of a PPO? It's still rolling along, but guess what? Some time ago they said that they may raise the withhold for "certain physicians." But this time we've told them up front that we'll get out if they do that to us. So far nothing has happened.

The point here is that you, as a practice, *can* have some influence on these large corporate decisions. If it's a managed care plan, they need quality primary care physicians. You also have a lot of influence with your patients and their employers when they're looking at these different plans.

Conclusion.

If you read somewhere that your area is targeted for managed care plans, take the time to learn something about them. And don't hesitate to get some consultation. Physician management consultants can help. So can physicians who have been directors or medical directors of some plans.

These plans definitely need you, but you may or may not need them.

Plans for Those Without a Prayer

The under-insured, uninsured, and those otherwise disenfranchised medically—their ranks are growing, and they haven't a prayer of paying the price of admission to an HMO, a PPO, or any other of the managed care plans Dr. John A. Mapp talks about in his cautionary tale above. Dr. Mapp believes this situation must change, and he believes physicians must be "on the front end of the change, rather than just reacting to changes others have made." He urges physician support for the recommendations of his specialty society, the American Academy of Family Physicians; under these recommendations, all Americans not otherwise covered could be insured and have access to a broad range of essential health care services. For a copy of the AAFP Position Statement spelling out the particulars, write to the Virginia Academy of Family Physicians, 4211 Dover Road, Richmond VA 23221.

The three major components of the AAFP's recommendations are Medicare, employer-provider coverage, and a restructured Medicaid program. These access routes are key also to the American Medical Association's Health Access America program, which was unveiled in Washington earlier this year. The Medical Society of Virginia has endorsed this program and at the 1990 session of the General Assembly lobbied successfully for the passage of legislation dealing with Medicaid reform, basic coverage policies for certain small employers/groups, and research into mandated insurance benefits with a view to limitation. MSV President William H. Barney has said that the Society's legislative committees and Council will continue to work for implementation of the total AMA access package.

—A.G.

Caveat Signator: Evaluating the Fine Print

Does the contract offer you a fair exchange
for the fees, time, flexibility you give up?
An affiliation tilted toward the managed care plan
could do your practice more harm than good.

by Geoffrey Anders, JD, CPA

An increasing number of doctors are choosing to participate in Health Maintenance Organizations (HMOs), Preferred Provider Organizations (PPOs), and other managed care plans.

There are some good reasons for doctors to affiliate with managed care operations. In the right circumstances, they can provide your practice with an increased patient base and a steady flow of income. Yet participation with managed care plans is not right for every practice; and though one managed care plan is right for you, another may not be.

BUSINESS CONSIDERATIONS

Sometimes it is difficult to withstand the pressure, real or imagined, to join a managed care system. Hospitals often urge doctors to participate as a way of bolstering admissions. Specialists are concerned that non-participation may result in fewer or no referrals from participating primary physicians. Some managed care plans will themselves use tough tactics to get doctors to sign up, for example, threatening that the

opportunity is "now or never." Usually, however, the pressure passes and the competitive fears are shown to be overblown. Few medical communities are so dominated by just one or two managed care programs that they preclude lucrative private practice, or the introduction of competing plans.

Participating with managed care plans should involve trade-offs, though too often doctors give discounts or accept capitation risk with only vague promises of patient volume in exchange. You should compare carefully the concrete opportunities being offered with what the plan asks you to give, e.g., fee discounts or unlimited services. Test those opportunities against your practice's goals and characteristics.

Do you really need this plan? What will you gain compared to what you lose?

Look at the plan's actual subscriber base—does it give you access to a patient group that you would like to serve? If you are offering a reduced fee, will the plan guarantee you at least a minimum patient volume? Or can the discount be placed on a sliding scale, tied to volume?

Remember, too, you do not want to let any one plan become too large a share of your practice base. Fifteen percent is a good benchmark, beyond which you should move only carefully. When one plan controls too large a portion of your patient base, its decisions and financial well-being can affect your practice negatively.

THE CONTRACT

Geoffrey Anders is president of The Health Care Group, Inc., a practice management consulting firm. Address reprint requests to him at 140 West Germantown Pike, Suite 200, Plymouth Meeting, Pennsylvania 19462. Multicopy requests for distribution to groups will be honored. © 1990.

Once you have made a strategic decision to participate with a managed care plan, your attention must turn to the rules that will guide your participation: the contract. Each organization will develop a contract

designed to meet its individual goals; therefore, while the broad outlines of managed care contracts are similar, you cannot assume the details will be, too. An arrangement that looks quite beneficial to you on the surface can turn into a liability when you dig into the contractual obligations.

A contract is a legally binding agreement. Do not sign it unless you understand and accept both the rights and responsibilities created. Feel free to negotiate a key point with a plan. And remember, once an agreement is signed, the law will not release you from its terms because the contract proves to be disadvantageous.

Because managed care contracts can be quite different from other types of contracts a doctor comes into contact with, it is important to understand their subtleties. Here is a review of some key points about managed care contracts and some key questions to ask either yourself or the plan's representative. Keep in mind that this is a general overview. If you are considering signing a managed care contract you should have an experienced attorney review it in detail.

General Contractual Issues

Quite often a managed care plan will retain the right to amend the contract, without giving you a similar privilege. Determine if a contract can be amended by the plan, and how quickly you can get out if the amendment is not to your advantage. Does the required notice for your terminating the agreement mesh well with the notice you receive of a change in the plan's terms? In any event, having to wait longer than 90 days to terminate your participation with a plan, for whatever reason, will not serve your interests. In general, try to avoid a situation where the plan may make unilateral changes in the contract. Especially, do not allow the plan to change unilaterally the services you must provide.

Managed care contracts do not always contain all the pertinent in-

formation that affects you. If a contract refers to an "Operations Manual" or to other documents, review them before signing the contract. Make sure you understand the plan's options in amending the manual; frequently, a contract cannot be amended without your approval, while a manual can. This means you could have little recourse when a manual change affects you negatively.

Vague language in a contract can come back to haunt you, e.g., "in a timely manner" or "general administrative duties." Whenever possible, have such language removed or clearly defined.

Most plans will not let you assign your contractual responsibilities; therefore, you should have the option to leave the contract if the plan chooses to assign its part.

Many contracts warrant the quality of medical care to be provided. This vague provision serves no good purpose, except that it may allow a plan to sue you in a contract action if you or they are hit with a malpractice suit. Such a provision should be struck from the contract.

Plans will often want the use of your name for promoting its services; you should have the same right, and the contract should provide for mutual use of names for promoting services.

Capitation and Fees

For primary care specialists, a standard arrangement in many plans is for it to pay you a set fee for each patient who designates you as the primary physician. This "capitation fee" is the same whether a patient needs care once a week or once a year. You should determine if the capitation fee offered you is a fair one. To do this, take a sample of your own patient base—one that is demographically similar to the plan's patient base—and review the services you provided them in the last year or more; based on that information, get a ball-park figure on how you would fare if the capitation fee were applied to your existing patients.

Patients with pre-existing conditions may skew the actuarial assumptions underlying your capitation payments—and so could cost you a great deal of money. While the plan must accept these patients, determine if the contract provides concessions to you for treating them.

Be sure the contract includes a process for regular increases or re-computation of fees. It is important that these be tied to some objective outside scale, e.g., Medicare fees or a medical cost index, rather than simply left open to negotiation.

Billing

One "benefit" often touted by managed care plans is the ease of billing functions: reduced paperwork, no bad debt risk and no collection efforts needed. But this benefit can be illusory. In fact, you may find that your regular billing activities are simply shifted from post-service, when you bill your own patients, to pre-service—where your staff must ascertain patient eligibility, receive authorizations, or obtain precertifications. You must be sure that these business activities can be handled efficiently within your current operations; if not, do the changes necessary to take on the plan's requirements constitute a worthwhile investment for you?

There are other questions to ask about the billing process. Examine the claims-submission routine to determine if it is reasonable, and if it will mesh with your billing processes. Review the time limit for submitting your claims to the plan. A 30- or 60-day period is too short; look for a period of six to 12 months.

Find out how long the plan may take to make payments: Can you live with that? Will the plan pay you interest if payment turn-around exceeds the agreed time period? If the plan goes into default, will you still be required to see patients? And if so, for how long, and what remedy is available to you?

What happens if you deliver medically necessary but "non-covered" services: May the patient be billed and under what circumstances? And

if the patient demands services that are not covered or approved by the plan, may you bill the patient directly?

Services

Make sure you can provide each service required by the contract. If not, are you expected to arrange for those services to be provided? Can those services be subcontracted? Should the contract require it, are you willing to expand your services or facilities to provide the full range of services described?

Coverage

What coverage procedures are acceptable? If your current coverage doctors are not also plan participants, will they be reimbursed for their services by the plan?

Referrals

What happens if you need to refer outside the plan? Are there set procedures for approvals and reimbursements? Are you comfortable with the plan physicians to whom you likely would be required to refer?

Utilization Review

Occasionally, managed care contracts obligate you to serve in the plan's utilization review process. Determine the nature and frequency of those responsibilities—and if you really want to be involved in that process. If you will be serving, you should be indemnified by the plan against liability arising from your utilization review activities.

Be certain that you understand the utilization review process and determine if you find it a reasonable one. Is there formal education on the plan's utilization requirements? How are over-utilization issues handled: Are there provisions for negotiations and arbitration? Is there a "rehabilitation" routine or might you be "cut off" summarily? And how are medical necessity disputes handled?

Risk Pools and Holdbacks

Many plans will withhold a por-

tion of the capitation fee to create a risk pool, as insurance against greater-than-expected expenses. If those dollars are not needed to cover unexpected costs, they are shared with you. Several points about the risk pool holdback should be made clear in the contract: How is your contribution returned and when? Are you obligated to cover any deficit in the risk pool? If you terminate plan participation, do you still benefit from risk pool distributions for the period of your involvement?

Find out who else is included in your risk pool. Be sure you are comfortable with their practice style and with being financially tied to these persons.

Liability

In the past, plans often included an indemnification, or "Hold Harmless" clause, stating that the doctor was responsible for the costs from suit or claim in which the plan was involved. Today these clauses are usually found in watered down form, although you should still negotiate them out of the contract.

Be sure to send a copy of any indemnity or hold harmless language to your insurer. Insurers will not cover contractually assumed liability, and you will want written assurance from your malpractice carrier that you will be covered under the specific language of the plan's contract.

If your practice is incorporated, it—not an individual doctor—should be making the agreement with the plan. Therefore, sign the contract as a corporate representative, not on your personal behalf.

Protecting Your Future

Check the managed care plan's history in other parts of the country. You should know if there have been any serious problems between the plan and its doctors. It is especially important to know if the plan has a history of operating a staff or group model—employing its own doctors, rather than contracting with existing practices. If this is the case, the plan could simply be using you to get

itself up and running, planning to take away your patient flow and direct it in-house later.

To protect yourself, negotiate into the agreement a provision that the plan will not provide direct services during the term of your contract—and for one or two years after your agreement ends.

Retain your right to accept or reject an individual new patient; you may not want to handle a patient who has a history of non-compliance or of being litigious.

Medical Records

If the contract authorizes the plan to have access to the patients' records, what provision is made for your legal and ethical responsibilities regarding confidentiality? And if you are required to transmit patient records to the plan or another provider, who pays the expense?

Conclusion

It is essential for you to understand fully what you are obligating yourself to when signing a managed care contract. The plan may not make it easy for you; your obligations may be spread throughout the document—or through several documents in addition to the contract—and may be implied rather than given explicitly. Allowing an experienced attorney to review the contract is a wise course of action.

You must also consider the effects that a managed care plan's operating scheme may have on your practice's existing routines. Gaining plan patients may not be worth the effort to make over your practice's billing, scheduling and coverage procedures. A practice management consultant can give you a detailed picture of how a managed care affiliation will affect your business routines.

Finally, in evaluating a managed care contract, you must determine if you are getting a fair exchange for the fees, time, or flexibility you may be giving up. In the long run, an affiliation tilted toward the managed care plan could do your practice more harm than good.



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Ruptured Abdominal Aortic Aneurysm: The Case for Elective Resection

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ABDOMINAL aortic aneurysms (AAA) represent a relatively common finding in the general population, occurring in nearly 2% of consecutive postmortem examinations.¹ With an expanding senior citizenry, more attention should be directed to appropriate diagnosis and management of this degenerative complication of arteriosclerosis.

The term aneurysm is derived from the Greek "aneuryneim," meaning to widen or dilate. Once widening or dilatation of the aorta begins, the natural history of AAA is that of progressive dilatation leading ultimately to rupture.²

DuBost reported the first successful elective resection of an AAA in 1952.³ In 1953, Cooley and DeBakey reported repair of a ruptured AAA, a situation which previously had been universally fatal.⁴ Data reported by Estes in 1950 allowed comparative analysis by Crawford and others to define the positive impact of AAA repair on actuarial tables in the ensuing decades, documenting continued progress in the treatment of abdominal aortic aneurysms.^{5,9}

The diagnosis of AAA usually can be made by physical examination. In more than 80% of cases,³ a pulsatile periumbilical mass can be palpated.² The characteristic calcified rim outlining the aneurysm wall on IVP, plain abdominal, or back x-ray often locate heretofore unexpected AAA. Once suspected, more precise diagnostic definition of the aneurysm as well as accurate measurements can be obtained by scanning techniques such as ultrasound or CT scan. Angiography provides additional data when precise adjacent anatomical information is needed for surgical planning.

The clinical presentation of AAA follows no set pattern. Patients may complain of nonspecific abdominal, flank or back discomfort. Occasionally, gastro-

intestinal bleeding may occur due to aortoenteric fistula formation. Rarely, ureteral obstruction is encountered. Distal emboli secondary to fragmentation of luminal debris occurs, but rarely. Sudden thrombosis of AAA is described, which may be dramatic in presentation, but fortunately is unusual.

The relationship between size of AAA, symptoms and rupture deserves careful consideration. Szilagyi suggested that size of AAA and likelihood of rupture were related, and that AAA 6 cm or larger merited repair.⁶ Darling reported an autopsy series of patients with ruptured AAA and noted that a full 20% had AAA that were 4 cm or less.⁷ Thus a "small" aneurysm is not necessarily a safe aneurysm. Citing the natural history of AAA, Crawford has suggested consideration for repair as soon as the diagnosis of AAA is made regardless of size, unless severely compromised cardiopulmonary status or imminent death from malignant disease makes this approach unrealistic.⁹

Rupture of an AAA is a well-known catastrophe. The diagnosis is suspected by the triad of abdominal or back pain, a pulsatile abdominal mass, and shock. Although without surgery the mortality approaches 100%, the results with surgical intervention have been disappointing in most series.¹⁴ To define the treatment experience with ruptured AAA in the Norfolk area, a retrospective review was undertaken with particular attention to possible prognostic factors.

Materials and Methods

For the 13-year period 1975-1987, 81 patients operated on for ruptured AAA were identified. Seventy-one charts were available with adequate information for retrospective review. Thirty-three variables were reviewed as possible prognostic indicators, divided into seven categories, and submitted to multivariate analysis (Table 1). Cases were divided into two arbi-

Table 1. Prognostic Variables in 71 Patients with Ruptured Abdominal Aortic Aneurysms (35-92 yrs, mean age 70.3; 81.7% males; 88.7% Caucasians).

1. General: age*, sex, race, date of operation
2. Past Medical History: ASHD, CVA*, PVD*, DM, COPD, previous knowledge AAA
3. Presentation: pain, mass*, syncope*, systolic BP, arrest, shock*
4. Delay: onset of symptoms to ER*, ER to OR*
5. Intraoperative: OR time*, time of day*, extent of rupture, blood loss*, transfusions*
6. Perioperative Complications: respiratory insufficiency*, pneumonia, ARDS*, MI*, arrhythmia*, hemorrhage, coagulopathy*, renal failure, sepsis*
7. Hospital Stay: extended*

* = statistically significant

trary time groups (Group I January 1975-June 1981; Group II July 1981-December 1987) for comparative purposes (Table 2).

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Table 2. Mortality in 71 Patients with Ruptured Abdominal Aortic Aneurysms.

Date	Patients	Deaths	Mortality
Period I: Jan 75-June 81	23	12	52%
Period II: July 81-Dec 87	48	24	50%
Total, 1975-1981	71	36	50.7%
Group I: 1976-1981	188		6%
Group II: 1984-1986	172	5	2.9%

Results

Caucasian males in their 70s were the most common patient profile (Table 1).

Multivariate statistical analysis with stepwise regression of the 33 variables showed that 20 were significant (p value of 0.05, Table 1). None of the variables were significant in the general category except age.

In the past medical history category (PMH), only cerebrovascular accident (CVA) and peripheral vascular disease (PWD) were significant. Urgency prevented a detailed PMH, which may have made this category incomplete.

In the mode of presentation category, mass, syncope and shock were significant, whereas pain was not. Range of blood pressure was not significant.

In the delay category, both delay from onset of symptoms to emergency room presentation and delay from emergency room arrival to arrival in the operating room were significant.

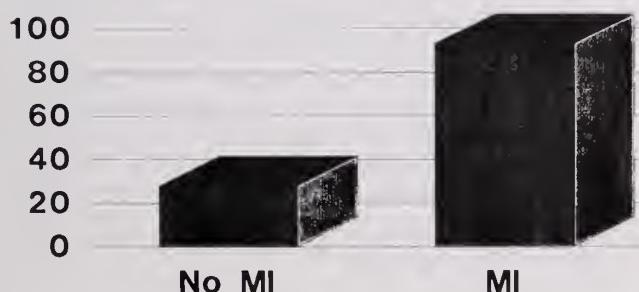
In the intraoperative category, blood loss and number of transfusions were significant as well as duration of operation and time of day (day vs night). Extent of rupture (free peritoneal vs confined to retroperitoneum) was not significant.

In the postoperative complication category, respiratory insufficiency, ARDS, MI, arrhythmia, coagulopathy, and sepsis were significant as well as extended hospitalization and renal failure. Particularly impressive was perioperative myocardial infarction, which carried a 93% mortality (Fig. 1).

Mortality for the earlier half of the study (Period I) was essentially the same as the later half (Period II), 52% vs 50%, with a combined mortality of 50.7% (Table 2). To compare this to elective cases, two

Fig. 1. Influence of Perioperative Myocardial Infarction on Mortality in Patients with AAA.

% Mortality



separate patient groups were reviewed. Group I (1976-1981), consisting of 188 patients, underwent elective aortic aneurysmectomy with a 6% mortality (Table 2). Concerned by this mortality, a protocol to maximize cardiopulmonary and general medical status was instituted for patients undergoing aortic surgery including use of intraoperative Swan-Ganz catheters in all patients, overnight preoperative monitoring in selected cases, and preoperative cardiac assessment via isotope scanning (persantine-thallium) with selective cardiac catheterization and coronary bypass surgery where appropriate. Group II patients (1984-1986; 172 patients), consisting of those patients prepared by this protocol, were found to have a 2.9% mortality following aortic surgery with no deaths for those done through the retroperitoneal approach.⁹ Although Group II patients represented a mixed group of patients with both abdominal aortic aneurysm and aortoiliac occlusive disease, the operative procedures and perioperative hemodynamics are basically comparable.⁹

Discussion

Mortality figures for patients operated on for ruptured AAA are high in most reported series. A survey of representative series of ruptured AAA over the past 25 years defines a mortality ranging from 37% to 65% (Table 3).⁹⁻¹⁵ The mortality for patients seen in the

Table 3. Comparison of AAA Mortality in Seven Reports 1964-1989.

Series	Deaths/Total	Mortality
Hume et al 1964 ¹⁵	10/27	37%
Shumaker et al 1973 ¹¹	87/158	55.1%
Hildebrand et al 1975 ¹⁰	67/131	51.5%
Ottlinger 1975 ¹³	58/100	58%
Chidichimo et al 1982 ¹²	28/65	43%
Moore et al 1984 ¹⁴	29/45	65%
Gregory et al 1989 ⁹	36/71	50.7%

Norfolk area shows a similar survival pattern and also shows that the mortality has changed very little over the past 13 years, remaining in the 50% range.

Careful analysis of multiple possible prognostic variables impacting on survival suggest that both the severity and duration of preoperative cardiovascular collapse had a significant effect on the outcome in this elderly group of patients. Furthermore, intraoperative factors have an additional devastating effect on these already compromised patients, leading to postoperative complications which frequently have a fatal result. Areas of possible beneficial change are difficult to identify and are clearly speculative, except to direct efforts towards control of hemorrhage and to minimize time in both the emergency room and the operating room.

In contrast to these disturbing mortality figures associated with ruptured AAA, mortality from elective aortic surgery fell dramatically during this same time

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period by carefully assessing and maximizing cardio-pulmonary factors preoperatively, as well as employing a more consistent monitoring program in the perioperative period.

In comparing the results of surgery for ruptured AAA, which have remained at 50% mortality for the past 13 years, versus the mortality for elective aortic surgery, which has fallen to 2.9% over the same time frame, the conclusion is obvious. Every possible effort should be made to identify patients with AAA, consider elective repair, and avoid the emergency of rupture AAA and the attendant 50% surgical mortality.

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ATLL Complicated by Strongyloidiasis and Isosporiasis: Case Report

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WE PRESENT a patient of Japanese origin whose persistent gastrointestinal symptoms were initially diagnosed as indicative of strongyloidiasis, but whose subsequent course led to the diagnosis of HTLV-I-associated adult T-cell leukemia/lymphoma (ATLL). Although the association of HTLV-I and *Strongyloides* infection has been well described, this to our knowledge is the first reported case of both isosporiasis and strongyloidiasis complicating ATLL.

Case Report

A 52-year-old Japanese female without significant past medical history presented with complaints of mild epigastric pain, anorexia, weight loss, and diarrhea in July, 1987. Evaluation of the patient's stool revealed infection with *Strongyloides stercoralis*, and thiabendazole therapy was initiated. Despite adequate therapy for strongyloidiasis, the patient continued to have vague abdominal discomfort, mild nausea, occasional episodes of loose stools, and lost 20% of her total body weight within three weeks. She was admitted to her local hospital for parenteral nutrition and diagnostic studies. Ultrasound and CT scans of the abdomen and pelvis were unremarkable. Colonoscopy and air contrast barium enema revealed many superficial ulcerations in the rectosigmoid, with the remainder of the colon and terminal ileum being normal. Biopsy of the colon was consistent with a nonspecific colitis. The patient was discharged after a significant weight gain and clinical improvement.

Three weeks after discharge the patient was seen

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with complaints of ear pain, dry mouth, dysgeusia, and dysosmia. Physical exam at this time revealed several firm submandibular lymph nodes and biopsy of a minor salivary gland reportedly showed an atypical lymphocytic infiltrate. The patient was again having abdominal discomfort and anorexia and was readmitted for further evaluation in October, 1987. Physical examination revealed a cachectic, ill-appearing woman with bibasilar rales on lung exam, hepatomegaly, and diffuse lymphadenopathy. Significant laboratory data included SGOT 70 U/I (nl 5-35 U/I), LDH 1529 U/I (nl 297-537 U/I), alkaline phosphatase 521 U/I (nl 43-122U/I) and albumin 1.9 gm/dl (nl 3.0-5.2). CBC and other chemistries were within normal limits. Stool examinations for ova and parasites were negative at this time.

Extensive evaluation included repeat abdominal ultrasound and CT scan which showed ascites, intrahepatic and extrahepatic ductal dilatation, hepatomegaly and enlarged retroperitoneal nodes. A chest CT scan showed multiple, superior mediastinal and axillary nodes, a small pericardial effusion, and bilateral pleural effusions. Bone marrow biopsy was reported as normocellular. Liver biopsy revealed an atypical lymphoid infiltrate. Percutaneous biopsy of the liver showed a mixed portal cell infiltration suggestive of acute and chronic cholangitis with a focus of atypical cells. Thoracentesis gave evidence of atypical lymphocytes. Periaortic lymph node biopsy also demonstrated a polymorphous lymphocytic infiltrate.

One week after admission, a follow-up stool examination revealed *Strongyloides* larvae. The patient was once again treated with thiabendazole and parenteral nutrition. She developed fever and clinical deterioration two weeks into her hospital stay. Cultures of blood, urine and pleural fluid were negative. Laboratory data at this time included WBC 150,000/mm³ (28% segmented neutrophils, 51% lymphocytes, 15% immature cells and 1 nucleated RBC), hematocrit 42%, platelets 217,000/mm³, alkaline phosphatase 536 U/I, LDH 5459 U/I, and calcium 12.4 mg/dl. Peripheral blood smear was thought to exhibit a leukemoid reaction with bizarre monocytoid cells. Repeat bone marrow biopsy revealed a slightly hypercellular marrow with polymorphous lymphocytic infiltrates and nuclear atypia.

The patient was then transferred to the University of Virginia Hospital late October 1987. On presentation, she was an anxious woman in moderate respiratory distress. Vital signs included a temperature of 38.8°C, a blood pressure of 100/60, a pulse of 140, and labored respirations of 30 per minute. Her skin was clear. Mucous membranes were dry. Her neck was supple with marked jugular venous distention. Enlarged cervical, submandibular, axillary, and inguinal lymph nodes were present. Diffuse ronchi were heard with diminished breath sounds at both bases. Cardiac examination was remarkable for an S3 gallop. Abdominal

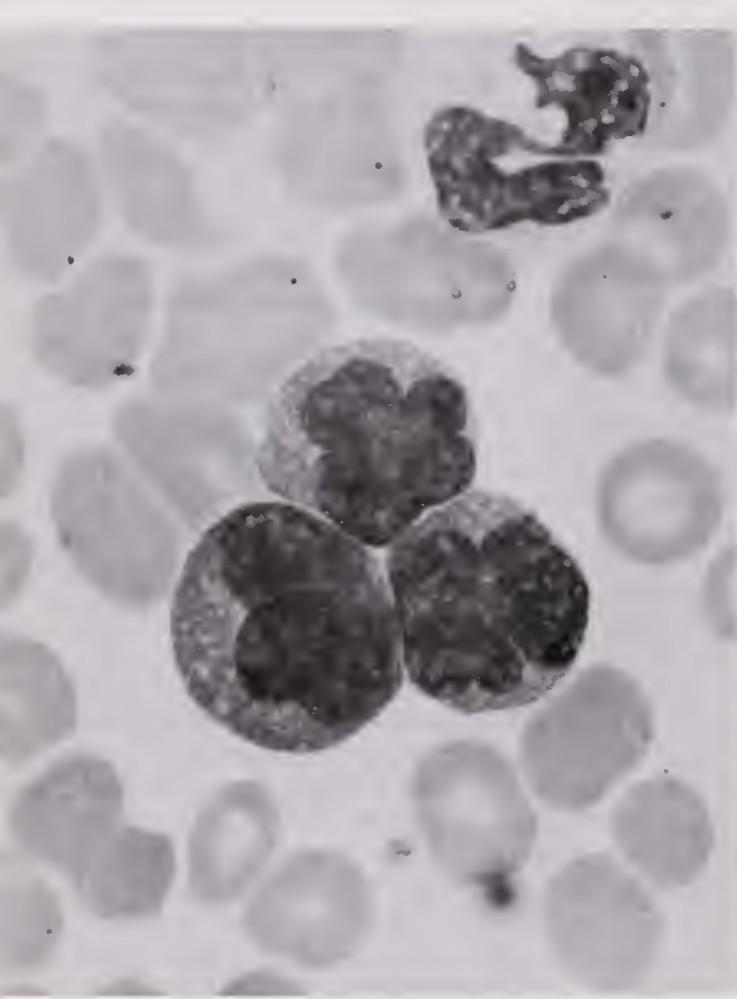


Fig. 1. Peripheral blood smear in patient with HTLV-I-associated adult T-cell lymphoma demonstrating characteristic lymphocytes with multilobulated nuclei (1250x).

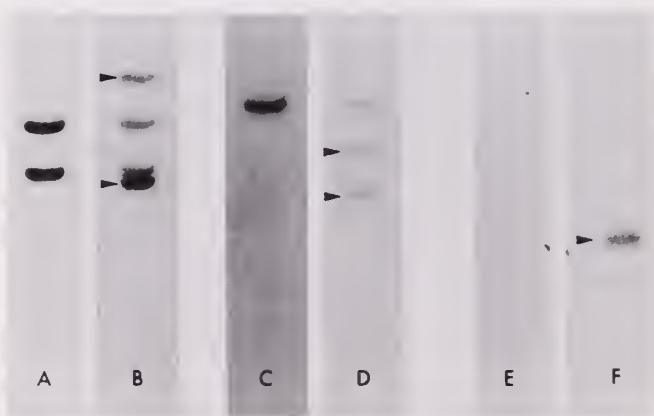


Fig. 2. Southern blot autoradiogram of control DNA (lanes A,C); HTLV-I negative T-cell lymphoma DNA (lane E); and patient DNA (lanes B,D,F). Probes included the T cell receptor gamma chain joining genes (lanes A,B); beta chain constant gene (lanes C,D); and the polymerase gene of HTLV-I (lanes E,F). Rearranged clonal bands are indicated by arrows. DNA was digested with the restriction enzymes BamHI (lanes A-D) or EcoRI (lanes E,F). (Reprinted with permission of the American Society of Clinical Pathologists from the *American Journal of Clinical Pathology*).

exam revealed diminished bowel sounds, a liver span of twenty-three centimeters, a palpable spleen tip, and diffuse, mild tenderness. Extremity and neurologic examinations were unremarkable.

Pertinent laboratory data included WBC 220,000/mm³ (25% segmented neutrophils, 7% monocytes, 1% lymphocytes, and 67% large mononuclear cells with multilobulated nuclei), hematocrit 39%, platelets 109,000/mm³, calcium 12.0 mg/dl, albumin 3.2 gm/dl, alkaline phosphatase 525 U/l, SGOT 171 U/l, SGPT 35 U/l, LDH 3939 U/l. An arterial blood gas on 2 liters of nasal oxygen revealed pH 7.44, pCO₂ 25 mm Hg and pO₂ 54 mm Hg. Chest X-ray was notable for bilateral pleural effusions and diffuse interstitial markings. Head CT scan was normal. Cerebrospinal fluid cytology was negative.

Immediate review of all previous pathologic specimens confirmed that the atypical infiltrates seen in the biopsies of the bone marrow, colon, salivary gland, lip, liver, periaortic node, as well as the atypical cells seen in the pleural fluid and peripheral blood were similar (Figure 1). Lymphocyte marker studies of peripheral blood revealed monoexpression of T helper cells (CD4 93%, CD3 93%, CD5 94%, CD2 96%, CD8 1%, CD7 1%, CD19 0% HLA-DR 50%, and Leu8 48%; the HLA-DR and Leu 8 positive cells indicate activation antigens on some of the tumor cells; the lack of CD 7 marker demonstrates aberrant expression of T cell monoclonal antigens which is typical for T cell neoplasms). This clinical presentation was consistent with adult T-cell leukemia/lymphoma. This was supported by a positive EIA for HTLV-I antibody (performed by Specialty Laboratories, Inc., Los Angeles CA). HIV antibody titers were negative. Southern blot analysis revealed clonal rearrangement of the T cell receptor gamma and beta genes consistent with a monoclonal T cell neoplasm. Hybridization with the HTLV-I polymerase gene probe confirmed proviral integration (Figure 2).

Additionally, stool samples revealed the presence of *Isospora belli* oocysts (Figure 3). There were no *Strongyloides* larvae found and thiabendazole was discontinued as she had received a dosing schedule of 25 mg/kg for five days. The isosporiasis was treated with trimethoprim (160 mg) and sulfamethoxazole (800 mg) four times a day for ten days and then twice a day for a total of three weeks. Stool specimens were remarkable for heavy shedding of oocysts two days into therapy with none present after four days.

The patient's leukemia/lymphoma was treated with cyclophosphamide, doxorubicin, vincristine, prednisone, and prophylactic intrathecal methotrexate. She had an initial dramatic response with disappearance of the pleural effusions, peripheral lymphadenopathy, and hepatosplenomegaly. In mid-November, her WBC was 9,700/mm³ without evidence of leukemic cells on peripheral blood smear.

In December 1987, however, her WBC increased to

30,000/mm³ with relapse of her ATLL. Recurrent infection with both *Isospora* and *Strongyloides* was treated with thiabendazole 750 mg bid for 3 weeks and trimethoprim-sulfamethoxazole (320/1600) twice daily for ten days, then decreased to half this dose. Her clinical condition deteriorated over the next month with development of increasing tumor burden, respiratory failure, and eventual sepsis. The patient expired January 1988. Permission for postmortem examination was not obtained.

Discussion

This case is a classic presentation for adult T-cell leukemia/lymphoma, first described in Japan in 1977 as an aggressive proliferation of abnormal lymphocytes characterized by hypercalcemia, widespread lymphadenopathy, skin lesions, hepatomegaly, splenomegaly, and variable respiratory, gastrointestinal and central nervous system involvement.¹⁻⁶ Although the patient had been in the United States for 15 years, she was originally from southern Japan, an endemic region where antibodies to HTLV-I are found in up to one-third of the population age 30 or older. Patients often experience an asymptomatic latent period of years before the abrupt and aggressive proliferation of malignant lymphocytes occurs.²⁻⁵ The high mortality rate in these patients results from the rapid, extensive dissemination of leukemic cells or from overwhelming opportunistic infections.

Both HTLV-I and HIV infections have been associated with malignancy, and both predispose to opportunistic infections. HIV has been shown to have a cytopathic effect on T4 helper cells. Alternatively, HTLV-I causes proliferation of the same population of lymphocytes. Exogenous infection of OKT4+ T cells with HTLV-I occurs and most of the lymphocytes do not undergo malignant transformation, although their immunologic function is impaired. In vitro studies show suppressive effects of HTLV-I infection on T helper, direct cytotoxic and natural killer cell function, although the mechanism of this suppression has yet to be elucidated.⁷⁻⁹ Thus, as seen in our patient, opportunistic infection may precede the development of malignancy.^{2-4,10,11} The HTLV-I provirus integrates randomly into the cellular genome and malignant transformation may occur in one of these cells with subsequent clonal proliferation, but this may take years, or may in fact never occur.²⁻⁴ The presence of a single band on this patient's Southern blot analysis indicates a single integration site in this monoclonal tumor population (Figure 2).^{12,13}

Opportunistic infections previously described in association with ATLL include *Pneumocystis carinii*, *Cryptococcus*, *Candida*, Cytomegalovirus, *Herpes simplex* virus, histoplasmosis, and as seen in this patient, *Strongyloides stercoralis*.¹⁴ *Strongyloides stercoralis* is an intestinal nematode which can pose a major threat to the immunocompromised host due to



Fig. 3A (above), Phase contrast and Fig. 3B (below), modified Kinyoun stain of *Isospora belli* containing two sporoblasts isolated from patient's stool. Organism averages 15 × 25 μM in size.



its propensity for hyperinfection and dissemination in this setting, its frequent association with gram negative bacterial sepsis, and because it is often not considered initially in the differential diagnosis of such patients.¹⁵⁻¹⁸ The association of HTLV-I and HIV with strongyloidiasis has been described, and with early recognition the initial infection and recurrences can be treated effectively.^{9,10,13} In the normal host, treatment with thiabendazole (25mg/kg) for two days is recommended. Immunocompromised patients are usually treated for 5-7 days, and recurrent infections may necessitate chronic suppressive therapy. Patients should also be retreated prior to receiving steroids or chemotherapeutic agents.^{15,16,18}

During the acute phase of this patient's malignancy, *Isospora belli* oocysts were found in her stool. Coccidial infections, which include *Isospora* spp. and *Cryptosporidium*, have been important clinically in association with AIDS and can be the presenting opportunistic infection of this syndrome, especially among Haitians.¹⁹⁻²³ There have been only two reported cases of *Isospora belli* infection in patients with ATLL.²⁴ Diagnosis is made by identifying immature and sporulated oocysts in stool by wet mount or modified acid-fast stain (Figure 3). Duodenal aspirates and small bowel biopsy may also be used for diagnosis.¹⁹⁻²¹ Eosinophilia may be present, but as with *Strongyloides*, its absence does not exclude the diagnosis. Fecal leukocytes are usually not present. Also of note, one patient with AIDS at autopsy was found to have *Isospora belli* in mesenteric and tracheobronchial lymph nodes as evidence for dissemination.²²

Our patient was treated with trimethoprim-sulfamethoxazole (160/800 mg) four times daily for ten days then twice a day for three weeks as previously recommended.¹⁹ There is some thought that this may be excessive in that symptoms resolve in 2-3 days.¹⁹ This patient was found to have a massive shedding of oocysts in her stool within 48 hours of therapy with subsequent negative stool examinations. Metronidazole and pyrimethamine plus sulfadiazine have also been successful in treating isosporiasis in small numbers of patients.¹⁹ However, at least 50% of AIDS patients have recurrent *Isospora* infection within a short time. Therefore, prophylaxis with sulfadoxine (25 mg) and pyrimethamine (500 mg) weekly, or trimethoprim-sulfamethoxazole (160/800 mg) three times a week has recently been shown to be effective.²³

Concurrent infections with other enteric pathogens can occur with *Isospora* infection. Approximately 5% of Haitian patients with AIDS and *Isospora* have been infected with either *Strongyloides*, *Giardia*, *Ascaris* or *Cryptosporidium*.¹⁹ Multiple enteric pathogens have also been reported in a patient with HTLV-I infection.¹⁴

Summary

In summary, we present this as the first reported case of both isosporiasis and strongyloidiasis complicating HTLV-I-associated ATLL. Prompt diagnosis and treatment of these parasites in the immunocompromised host are necessary to prevent severe wasting and dehydration. This should also prevent the significant morbidity and mortality associated with dissemination well-described for *Strongyloides*, and recently seen at autopsy in a patient with isosporiasis. Recurrent infections are common with both organisms, therefore chronic suppressive therapy and prophylactic treatment prior to chemotherapy or steroid administration is warranted.

Dr. M. Yoshida, Cancer Institute, Tokyo, kindly provided the HTLV-I hybridization probe, and Dr. Donald J. Innes, Department of Pathology, University of Virginia Hospital, kindly provided the photograph of peripheral blood smear.

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Two new solutions for making ticks "lose interest" so that they may be removed easily with head intact have surfaced in our household recently. Betadine® has been our standby for years, but when we were not able to find any recently, both Original Palmolive Dishwashing Liquid (biodegradable, contains no phosphorus) and a Tucks pad have allowed ticks to be removed after they had embedded themselves, with their heads intact.

Edward G. Davis, Jr., MD

15 Buck Branch Drive
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ABSTRACTS

These abstracts derive from the annual meeting of the Virginia Society of Otolaryngology/Head and Neck Surgery in Alexandria April 19-21. Dr. Steven A. Davie and Dr. R. Jack Eastham presided.

Acoustic Neuroma: Diagnostic Techniques.

Fred T. Shaia, MD, and Nancy L. Schay, CCC/A, Richmond.

In the past, the diagnosis of acoustic neuromas required a battery of audiological tests and invasive radiology techniques. It is now possible to limit the testing to two studies, an auditory brainstem evoked potential, the second a magnetic scan with enhancement. A patient with any one of the following should have an auditory brainstem evoked potential: 1) unilateral sensorineural hearing loss; 2) asymmetrical sensorineural hearing loss; 3) unilateral tinnitus; 4) dizziness. When the auditory evoked potential is abnormal in any of these situations, the physician is advised to proceed with a magnetic scan with enhancement. The auditory evoked potential is 98% reliable in detecting acoustic neuromas and should be the first and only test after an audiogram that is necessary for the diagnosis. If for some reason, such as a total sensorineural hearing loss, the brainstem study cannot be performed, then the magnetic scan with enhancement should be the required testing. With this format, the diagnosis of small acoustic neuromas and surgery with preservation of hearing is a genuine and reasonable goal.

Adenotonsillectomy in Children with Sickle Cell Disease.

Craig S. Derkay, MD, James R. Thomsen, MD, Gregory J. Milmoe, MD, Gordon Bray, MD, and Kenneth M. Grundfast, MD, Norfolk.

The pediatric patient with sickle cell disease is at increased risk during adenotonsillectomy under general anesthesia because of the possible development of a vaso-occlusive episode. Although the indications for adenotonsillectomy may be similar to those in patients without hemoglobinopathies, careful pre-operative planning in collaboration with the hematologist is mandatory. This report reviews management for 10 children, ranging in age 4-14, with sickle hemoglobinopathies who had adenotonsillectomy. Indications for surgery were recurrent streptococcal infections in 4 and obstructive sleep apnea in 6 of these children. There were no complications resulting from any of these procedures and the mean duration of hospitalization post-operatively was 2.0 days. The principal feature of pre-operative management was the administration of red blood cell transfusions designed to suppress the patient's endogenous erythropoiesis and

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reduce the concentration of sickle hemoglobin to less than 30%. During surgery, patients were kept hydrated with intravenous lactated ringers at 1.5-2.0 maintenance while oxygen saturation was continuously monitored with a trans-cutaneous oxygen monitor.

Our experience indicates that adenotonsillectomy can be performed safely in children with sickle hemoglobinopathies.

Oncocytic Malignancy of the Parotid Gland. Richard L. Scher, MD, Philip S. Feldman, MD, and Paul R. Lambert, MD, Charlottesville.

Oncocytic malignancy of parotid gland origin is a very rare neoplasm. A combination of cytologic, histologic, histochemical and ultrastructural evaluation is necessary to differentiate this salivary gland malignancy from other parotid neoplasms with cells exhibiting oncocytic features. Since 1984 we have diagnosed and treated 4 patients with oncocytic carcinoma originating in the parotid gland. Two of these patients presented with facial paralysis as their initial symptom, with all cases demonstrating extensive perineural invasion in the pathologic specimen. An initial diagnosis of oncocytic malignancy was correctly made pre-operatively in each case with cytologic analysis obtained by fine-needle aspiration. These patients have been treated with aggressive surgical resection followed with external beam radiotherapy, with all patients presently alive 3-56 months after diagnosis. This report will discuss the clinical and pathologic features of this rare malignancy, and by reviewing these and previously reported cases, suggest rational treatment options based on the biologic behavior of these tumors.

Angioedema of the Head and Neck Associated with Angiotensin Converting Enzyme Inhibitors. John F. Huhn, MD, Timothy R. Jones, MD, Marc A. Seftel, MD, W. Copley McLean, MD, and Carlos R. Ayers, MD, Charlottesville.

Angioedema of the head and neck is a potentially life-threatening condition of uncertain etiology. Recent experience of 11 patients with angioedema associated with angiotensin converting enzyme (ACE) inhibitors was reviewed to provide guidelines for appropriate diagnosis and management of this condition. Analysis of patient data revealed a median onset of symptoms 30 days after initiation of ACE inhibitor therapy with a range of 2 days to 10 months. Hospitalization was required in 6 patients with a mean stay of 2 days. Four cases are discussed to illustrate the broad range of clinical presentation, including severe respiratory distress. Treatment recommendations include discontinuation of any ACE inhibitor, airway maintenance measures, steroid administration, and control of hypertension with an alternative medication.

Definitive Emergency Laryngectomy. Michael A. Seichnaydre, MD, Anthony W. Jackson, MD, and Mary Ann Smith Frable, MD, Richmond.

Three tumor patients presented at the Medical College of Virginia Hospitals, Virginia Commonwealth University, with life threatening complications. Two patients had airway obstruction from large laryngeal neoplasms and one had hemorrhage from a tumor arising at the base of the tongue. The hemorrhage could not be controlled by medical means. All patients underwent emergency laryngectomy. The authors review all three cases and discuss the pros and cons of their surgical approach.

Audiologic and Electrophysiologic Findings in Patients with Benign Intracranial Hypertension. Richard Calleari, MD, Tucker Cook, CCC/A, Susan Fowler, CCC/A, and Aristides Sismanis, MD, Richmond.

We have previously reported that pulsatile tinnitus and hearing loss can be the major or only manifestations of benign intracranial hypertension (BIH) syndrome. Between 1981-89 31 patients with BIH syndrome associated with otologic symptoms have been diagnosed at the Medical College of Virginia. A prospective evaluation with audiometric testing consisting of puretone (air and bone conduction), speech discrimination, tympanometry and auditory evoked responses was carried out. The results of this prospective study are to be reported in this communication. We have strong evidence that intracranial hypertension can have a direct or indirect effect on the auditory system resulting in hearing loss. The pathophysiology of these processes are to be presented in detail.

Office Computerization: Benefits and Pitfalls. Charles M. Johnson III, MD, Charlottesville, and Steven A. Davie, MD, Blacksburg.

This discussion will consider the use of both the micro- and mini-computer in the organization of the medical office, particularly for otolaryngologists. It will include what these computers can do, what they can not do, and what one can reasonably expect an adequate software program to accomplish. It will also discuss what the practicing otolaryngologists should be looking for in any company selling medical software systems. This report is a direct outgrowth of the Computer Committee of the Virginia Society of Otolaryngology.

Surgical Management of Massive Parapharyngeal Space Neurofibroma. Michael Stamm, MD, and Anthony W. Jackson, MD, Richmond.

Neurogenic tumors are the second most common primary tumors of the parapharyngeal space. These tumors are usually benign and slow growing. Symptoms are produced by pressure on neighboring structures. These neoplasms expand in the directions of least resistance. Eventual mandibular displacement

and deformity can occur secondary to parapharyngeal space neurofibroma that is allowed to progress. The authors will discuss the anatomy of the parapharyngeal space along with the most common neoplasms found in this area. We will then review the genetics and pathology of neurofibromatosis and report about a case of massive plexiform neurofibroma of the parapharyngeal space treated at our institution. We recommend surgical removal of these neoplasms prior to massive growth that causes bony deformities and airway compromise.

Lymphoma of the Temporal Bone. Debara L. Tucci, MD, John F. Huhn, MD, and Paul R. Lambert, MD, Charlottesville.

Primary temporal bone lymphoma is a rare clinical entity. The single previously reported case describes a patient who presented with progressive facial weakness 2 years following surgical resection and radiotherapy for a lymphoma of the parotid gland. We report on two patients with primary temporal bone lymphoma, both of whom presented with facial paralysis. One patient, an elderly man, demonstrated complete recovery of paralysis after the first course of chemotherapy. One year after initial presentation this patient has extensive metastatic disease without current evidence of persistent disease in the temporal bone. The other patient, a 5-year-old boy, has shown no improvement in facial function 3 months after initiation of treatment.

Pediatric Otolaryngology Procedures in the United States 1977-1987. M. J. Kelleher, MD, and C. S. Derkay, MD, Norfolk.

Utilizing data provided in the National Health Survey, common pediatric otolaryngology procedures for the year 1977 through 1987 were analyzed. Uniform data was available for adenotonsillar surgery and myringotomy with insertion of ventilation tubes. Parameters examined included: total procedures, rates of procedure per 100,000 population, differences in sex, age and race of patients, changes in length of hospital stay, variations in surgical rates based on size of the hospitals, and regional variations in surgical rates and length of stay. Although adenotonsillar surgery remained the most frequent surgical procedure performed on children under 15 years of age, the total number of procedures and the rate per 100,000 population declined by over 50%. In the 10 years studied, tonsillectomies decreased from 414,000 to 197,000 total procedures in the age group less than 15 years and from 291 per 100,000 to 134 per 100,000 children. A similar decline was noted in the rate and numbers of myringotomy and tube insertions. A review of the available literature on past and present numbers of otolaryngic procedures is presented. Potential explanations for and implications of these declining figures are discussed including the effects of institutionalized

guidelines, reimbursement pressures, increasing manpower and changing venues in which these procedures are performed.

The Medical College of Virginia Smell and Taste Clinic: A Preliminary Report. M. G. Mellis, MD, A. Sismanis, MD, R. J. Delorenzo, MD, W. W. Campbell, MD, P. G. Heywood, MS, and R. M. Costanzo, PhD, Richmond.

Recent advances in the pathophysiology of chemosensory function have led to renewed interest in the treatment of patients with smell and taste disorders. We report the preliminary findings of such patients seen during the first year of the newly established MCV Smell and Taste Clinic. Patients underwent a complete otolaryngologic examination including nasal endoscopy and special olfactory function testing. In approximately one-third of the patients, we were able to establish a rhinologic cause responsible for their olfactory dysfunction. Topical intranasal steroids provided symptomatic benefit in many of these patients. Patients treated surgically had excellent results verified by improved scores on the MCV olfactory function test. We recommend that patients with smell and taste disorders undergo thorough evaluation including nasal endoscopy and formal chemosensory (smell and taste) function testing. Greater physician awareness of recent advances and the availability of specialized diagnostic tests will lead to improved diagnosis and treatment of patients with smell and taste disorders.

Head and Neck Reconstruction Using Surgical Flaps: Classification, Clinical Considerations, and Pertinent Physiology. Michael F. Pratt, MD, and Sherard Tatum, MD, Norfolk.

Over the years the use of flap reconstruction in head and neck surgery has become commonplace. There have been many recent advances in the understanding and classification of these flaps. They have enhanced the rehabilitation of cancer and trauma patients by permitting immediate resurfacing of large skin and mucosal wounds, carotid artery protection, immediate restoration of near-normal physiology, and complicated multi-staged reconstructive techniques. This presentation will include a classification of flaps commonly used in head and neck reconstruction based upon a scientific understanding of vascular anatomy and a discussion of basic clinical considerations in flap design and flap physiology. Many forms of therapy have been used in recent years to improve tissue survival in skin flaps. Most are focused on improving tissue oxygenation or decreasing metabolic needs. An outline of new work in our laboratories designed to investigate the pharmacologic enhancement of random skin flaps will be presented.

VIRGINIA MEDICAL

No Smoking

AN earlier article in VIRGINIA MEDICAL (November, 1988) queried hospitals in Virginia in regard to smoking on hospital premises. A questionnaire was mailed to 115 hospitals and of the 97 responding only one banned smoking entirely. Four allowed complete freedom to smoke and the remaining 92 restricted smoking in various ways. An editorial in the same issue suggested a gradual approach to the elimination of smoking, with the first step being restriction of smoking everywhere except for a single designated area.

It appears that a modification of this approach has been adopted. Last fall the Virginia Hospital Association's Board of Directors called for member hospitals to adopt smoke-free policies by January 1, 1990, and to

implement a full-scale smoking policy by January 1, 1991. The VHA defines a smoke-free facility as one which "bans smoking everywhere, except in substance-abuse or psychiatric wards. Patients are allowed to smoke only in private rooms. A smoke-free facility also phases out designated smoking areas for staff and visitors."

A recent issue of *Airways*, a publication of the American Lung Association of Virginia, lists 16 Virginia hospitals as smoke-free under the definition for 1990. Nineteen more plan to become smoke-free by January 1, 1991. As this was being written a communication arrived from the Medical College of Virginia, announcing inception of a smoke-free policy as of the first of May 1990.

E.L.K., JR.

Lies and Cynicism

THE LONG RIDE to Richmond is only partially alleviated by listening to public radio stations, but this program was much more interesting than usual. The interviewee was the author of a recent book on the current level of public cynicism. The question under discussion was, of course, how much more cynical had the public become about politicians, government, big business and life in general.

Both interviewer and interviewee agreed that recent events had definitely had a negative effect upon the public: negative political campaigning, congressional ethics or the apparent lack thereof, executive branch sleaziness in pursuit of personal gain, big oil's lack of preparation for big oil spills in spite of multiple assurances before the fact, insider trading on the stock

market, bending the rules to make \$500 million dollars a year, etc.

Yes, the average American's trust in government, business and his fellow man had fallen dramatically. Cheating and lying were expected, anything to get ahead was the norm, manual labor was in disrepute, and cynicism was rife in the land.

Listening to this negative vein of thought, I considered that things were better in our rural area. In the mountains of Patrick County around my farm the people still value honesty, integrity, hard work, honor, duty, country.

My good friend, Wallace, is one of those traditional people. He voluntarily served two terms in the military forces. He personally built his modest home with help

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only from his wife and two small boys. In his many past vocations he had served as an officer of the law, had farmed, and had retired only when totally disabled.

Many times Wallace has told me, "Doc, I always tell the truth. You can't catch a man in a lie if he is telling the truth."

But then he pauses and adds, "Of course, if I'm losing the argument, I might have to lie just a little."

H. S. CAMPELL, MD

Medical Research Funding

GRANT funding for medical research is lagging. The Administration's request of \$7.930 billion for the National Institutes of Health for FY 1991 does not provide sufficient funds to fully support the current level of effort. Funding for research grants in 1989 provided sufficient monies for only 29 percent of those applications recommended for funding. The FY 1991 funding rate for new and renewal research grants will result in an all-time low.

The Ad Hoc Group for Medical Research Funding, composed of approximately 140 American research organizations and including the Association of American Medical Colleges, recommends an increase to \$9.237 billion. Such an increase has the potential of adding millions of dollars for biomedical research and training to all three Virginia medical schools, as well as other graduate and research programs in the state. This added funding is badly needed. Write to your congressman.

E.L.K., JR.



Guest Speaker: The Physician as Manager, an Exhortation

WE LIVE in a period in which the demands on physicians continue to grow. These pressures include demands for more of our time, for more teaching, and for more perfection in practice. At the same time there are increasing problems with substance abuse, family discord, suicide, depression, and physician "burnout." While there may be a number of reasonable strategies for dealing with these demands, the principles of good management must have a prominent place in our approach to these challenges.

First of all, doctors need to know how to manage people. They should be managers, leaders, and teachers of residents, students, patients, families, nurses, and other health care workers. How can we successfully fill these roles? There is no substitute for personal interest. We must understand all kinds of people and their priorities. Optimal communication is important and is enhanced by explaining, when feasible, what your requests entail. Such explanations decrease the chance for error, increase the chance for adaptation when necessary, and are compliments to your coworkers. Remember that there are differences between effectiveness and popularity. We must also understand the differences between motivation and manipulation. We must avoid being domineering, intimidating, and arrogant. The best leaders, teachers, and managers are approachable, communicative, and enthusiastic. They praise good performances and build self-esteem. High self-esteem leads to high performance. Constructive feedback, adding positive comments when criticism is necessary, is another principle of good management.

Second, physicians should be experts in time management. We all complain that there is not enough time. However, the central issue is not how much time there is; the crucial aspect of time is how we manage it. After all, everyone has the same 24 hours in each day. Unfortunately, most of us manage time poorly. We all know the world is full of competent people with good intentions who don't accomplish as much as they should. The most common cause of this shortcoming is poor time management. Time is managed best by making lists, breaking down problems to achievable tasks, and assigning priorities. Most of us are very good at making lists but are inept at assigning priorities. In order to assign priorities properly, one must understand the difference between urgency and importance. Things that are urgent are pressing, demand-

ing and seemingly relentless. They seem to call for immediate action, usually to the exclusion of all else. Examples of urgency in the personal realm include hunger, sleep, and protection of our egos. Examples in the educational realm include artificial deadlines, assignments, reports, and tests. Examples of urgency in the patient care arena include focusing on minor details such as isolated lab values or getting caught up in performing a specific procedure like an intravenous line during a resuscitation while ignoring the patient's overall condition or needs such as a cervical spine injury. However, many things that are urgent are not all that important and many things that are important are not all that urgent. Things that are important are things that are significant, meaningful, substantial, and of long-term consequence. Examples in the personal realm include exercise, recreation, health, personal values, religion, and family. Examples of things that are important in education include learning general principles rather than isolated facts. Also important but often of little urgency is the need for good teaching. Examples of things that are very important in the medical realm, but often are not very urgent, are the feelings and dignity of patients and their families or consideration of their quality of life. There is an old adage that reminds us that trivial matters are handled promptly while important ones are often never handled at all.

To acquire the necessary management skills, which include negotiating techniques, public speaking, teaching techniques, delegating, and leadership skills, we must continuously educate ourselves. We must be observers and connoisseurs of good management practices, looking for examples and role models inside and outside of medicine. There are many educational opportunities in this area, such as books, tapes, seminars, and courses.

In conclusion, the principles of management will be valuable to us as we face the many demands on the physicians of the future. Utilizing these principles will help our profession to grow stronger, more vital and more effective instead of succumbing to the pressures and challenges we face.

CURTIS G. TRIBBLE, MD

Excerpted from Dr. Tribble's baccalaureate address to the University of Virginia School of Medicine delivered on 24 May 1989. Address correspondence to him at the Department of Surgery, Box 181, University of Virginia Health Sciences Center, Charlottesville VA 22908.

VIRGINIA MEDICAL OBITUARY

- **William M. Bickers, MD**, longtime obstetrician/gynecologist in both Richmond and the Far East; Medical College of Virginia, 1904; age 82; died May 5, 1990.
- **William Walker Butzner, Jr., MD**, retired Fredericksburg family practitioner; University of Virginia School of Medicine, 1935; age 78; died April 27, 1990, in Easton, Maryland. He had twice served as president of the Fredericksburg Area Medical Society.
- **David Samuel Garner, MD**, retired Roanoke family physician; Medical College of Virginia, 1933; age 91; died May 6, 1990. He had been president of the Roanoke Academy of Medicine and of Blue Shield of Southwest Virginia.
- **Kermit Jackson Moore, MD**, family physician in Petersburg for 45 years; Medical College of Virginia, 1934; age 79; died April 15, 1990.
- **Marion D. Richmond, MD**, Martinsville pediatrician; Medical College of Virginia, 1959; age 57; died May 19, 1990.

Memoir of Frank Strickler 1908-1989

By Julian B. Doss, MD

Frank A. Strickler, MD, of Roanoke was born on July 12, 1908, and died September 9, 1989, at age 81.

Dr. Strickler grew up in Bridgewater, Virginia, and attended school there. He graduated from Bridgewater College, where was valedictorian of his class of 1928 and he was active in athletics. He then attended the University of Virginia School of Medicine, earning his MD degree in 1932. He served his residency in neuropsychiatry at the University of Virginia Hospital and continued his training at Stuart Circle Hospital in Richmond. As a staff member at New York City Hospital, he developed an osteomyelitis of the right upper arm that crippled and tormented him lifelong.

In 1942 he was staff member at St. Albans Hospital in Radford and a consultant in psychiatry at the Veterans Administration Hospital in Salem, Virginia. In 1943, Dr. Strickler established his private practice of neurology and psychiatry at the Medical Arts Building in Roanoke, Virginia continuing as a consultant at

the VA Hospital in Salem. He retired in 1982.

Dr. Strickler was among the pioneers in the field of neuropsychiatry as consultant and counselor. He was always friendly, kind and gentle. A real gentleman.

He was a member of the Church of the Bretheren, the Roanoke Valley Academy of Medicine, the American Psychiatric Association and The Medical Society of Virginia.

He is survived by his wife of 55 years, Mrs. Sylvia Shafer Strickler, and one brother, Stanley S. Strickler, both of Roanoke.

Memoir of Monford Custer 1914-1989

Dr. Monford D. Custer, Jr., died on November 23, 1989, after a distinguished medical career and long-term service to the Winchester community. Born in Coshocton, Ohio, on December 5, 1914, he was graduated from the Lawrenceville School in 1932, Princeton University in 1936, and Columbia University School of Physicians and Surgeons in 1940. After an internship at Robert Packer Hospital in Sayre, Pennsylvania, he began a fellowship in general surgery at the Mayo Clinic and Foundation in Rochester, Minnesota. His period of study was interrupted by World War II, at which time he served as a captain in the United States Army in Louisville, Kentucky, and chief of surgery at an army hospital in Puerto Rico. Following his service time, he returned to the Mayo Clinic to complete his fellowship and obtain a Master of Science in surgery from the University of Minnesota.

He and his wife, Lucy, came to Winchester in 1947, and Dr. Custer began an illustrious medical career which continued through his retirement in 1981. He started on the medical staff of the Winchester Memorial Hospital, served on many committees and was elected to the presidency of the staff in 1963. He was certified by the American Board of Surgery in 1948, and became a fellow in the American College of Surgeons in 1949. He published many scientific articles and editorials in national surgical journals.

In addition to membership in the AMA and The Medical Society of Virginia, he was a member of the Priestley Society of the Mayo Clinic and the Virginia Surgical Society, of which he was president in 1968. He was vice president in 1970 of the Southern Surgical Association and was the secretary for five years of the Southern Society of Clinical Surgeons and its presi-

dent in 1975.

One of his proudest accomplishments was the realization of establishing in Winchester in the 1950s the Winchester Surgical Clinic. Initially a general surgery group of Drs. Boyd, Horgan, Miller and Custer, it has ultimately grown to a multispecialty surgical group of 15 professionals practicing general, orthopedic, cardiovascular, thoracic and urologic surgery. In recognition of his worthy efforts and appreciation for the time and energy he extended in initiating the formation and ensuring the success of the Clinic, the building it presently occupies was dedicated to Dr. Custer prior to his retirement in 1981 and is known as the Monford D. Custer, Jr., Building.

His community interests were widespread and far-reaching. He was a long-time member of Christ Episcopal Church and served as vestryman and subsequently as warden. He was a member of the Handley Library Board, and served as chairman of the Development Committee when the new addition was realized. Dr. Custer was an active member of the Winchester Parking Authority, and subsequently served as chairman during the period when the first downtown parking garage was established. His life-long interest in golf enabled him to serve as president of the Winchester Country Club and subsequently become a founder and president of the Carper's Valley Golf Club. In 1968 he received the Citizen of the Year Award from the Winchester-Frederick County Chamber of Commerce.

During the 1970s Dr. Custer volunteered for service on the S.S. HOPE Hospital Ship, and subsequently became a strong supporter of Project HOPE and its headquarters relocation in Millwood, Virginia.

A very significant impact on the Winchester community has been made by the generosity of the Custer family, mainly in the areas of medicine, nursing, education and recreation. Dr. Custer, as a trustee of the Gallagher Foundation and through his own personal generosity, was instrumental in supporting many individual projects at Winchester Medical Center and assisted in establishing the Free Medical Clinic in its refurbished building. As a trustee of the Shenandoah College and Conservatory of Music and a recipient of its Distinguished Service Award, Dr. Custer, along with Mrs. Custer, was pivotally instrumental in ensuring the restoration of the Old John Kerr Building and its conversion to the Eleanor Wade Custer School of Nursing, Arts Center and Community Education Facility. This accomplishment will have a long-lasting influence on education, nursing and medicine in this community.

Dr. Custer married Lucy (Tide) Ellen Lamb of Mount Vernon, Ohio, in 1938. They had three daughters, Ellen C. Morgan of Los Angeles, California; Susan C. Sanders of Savannah, Georgia; and Linda C. Russell of White Post, Virginia; and one son, Dr. Monford D. Custer III of the Scott & White Clinic in

Temple, Texas. He was an avid fisherman, testing the waters from Pakistan to Alaska with his son, as well as the local waters of the Shenandoah River with his grandsons. The community of Winchester and the medical profession have lost a truly generous man, a leader with vision and accomplishment, a caring physician, and a Christian family man who enjoyed every aspect of his life. May he rest in peace.

Submitted for publication by the physicians of the Winchester Surgical Clinic.

Memoir of Peter Pastore 1907-1989

*By George H. Williams, MD,
Kinloch Nelson, MD, and Elam Toone, Jr., MD*

Dr. Peter N. Pastore was born in Bluefield, West Virginia, on November 8, 1907, and died in Richmond, Virginia, on September 16, 1989.

He was graduated from Bluefield's Beaver High School in 1926, continued his education for two years at Bluefield College, then received a BA degree from the University of Richmond in 1930, where he was a member of Phi Beta Kappa. His medical degree was earned at the Medical College of Virginia in 1934, where he became a member of Alpha Omega Alpha, and where he completed his internship and residency in 1936. He trained as a fellow at the Mayo Clinic and received an MS, Otolaryngology, from the University of Minnesota in 1939. In 1942 he became the first professor and chairman of the Department of Otology, Rhinology, Laryngology, Audiology and Speech Pathology at the Medical College of Virginia and so served until his retirement in 1976. As emeritus professor, he maintained an active role in student and resident education.

Dr. Pastore was always a dedicated academician with a precise mind, a gentle touch, and a sincere interest in students, patients, and colleagues. He was a tenacious researcher with a driving desire for documentation of all events, whether medical, historical, or simple relaxation with family or friends. His camera was always within reach and ready for action. He had an interest in the McGuire Veterans Administration Medical Center and was chief of otolaryngology there until 1976, after which he served as director of continuing medical education for otolaryngology until his death. He also served on the faculty of the American Academy of Ophthalmology and Otolaryngology from 1951 until 1975. Dr. Pastore had more than 45 scholarly articles covering all aspects of otolaryngology and medicine in general.

Dr. Pastore was president of the Richmond Eye, Ear, Nose and Throat Society, 1945-46; president of

the Virginia Society of Ophthalmology and Otolaryngology, 1952-53; president of the International Association to Secretaries of Ophthalmological and Otolaryngological Societies, 1959-62; president of the Medical College of Virginia Alumni Society, 1961-62; president of the American Cancer Society, Richmond Unit, 1960-61; and president of the International College of Surgeons, Virginia Division, 1962-64. He was on numerous boards and a member of most of the major medical, surgical and otolaryngological societies.

Dr. Pastore had a strong commitment to the community and religious organizations. He was president of the Catholic Physicians Guild, 1962-64 and of the board of Benedictine High School, 1964-66, and at his death was a member of the advisory boards of Benedictine and the National Council for Christians and Jews. He was president of the Easter Seal Society of Richmond, 1967-70, and served as treasurer of that organization, 1980-85. He was on the boards of The Richmond Children's Aid Society, 1944-47, and the Virginia Society for Crippled Children and Adults, 1954-74. He received numerous honors and awards, including: Medical-Scroll, Algernon Sydney Sullivan Award in Recognition of Spiritual and Humanitarian Service, 1982; Outstanding Contributions to the Communicatively Handicapped in Virginia, 1980; Virginia

Cultural Laureate Society, Laureate of Virginia, 1986; Award for Long Years of Commitment and Service to the Lives of People with Disabilities, Easter Seal Society of Virginia, and the Presidential Medallion, Virginia Commonwealth University, 1987.

Dr. Pastore was founder of several funds and services. He was the first director and lecturer, Medicine and Medical Advances, 1983, Elderhostel. He established the Peter N. and Julia Pastore Professorship in Otolaryngology Fund through the Medical College of Virginia Foundation. In 1977 he founded the Pastore Otolaryngology and Medical Artifacts Historical Collection, Medical College of Virginia/Virginia Commonwealth University, Tompkins-McCaw Library Special Collections Archives and devoted much of his time and resources to the artifacts collection during his retirement years. It is a tribute to his desire to document and preserve history.

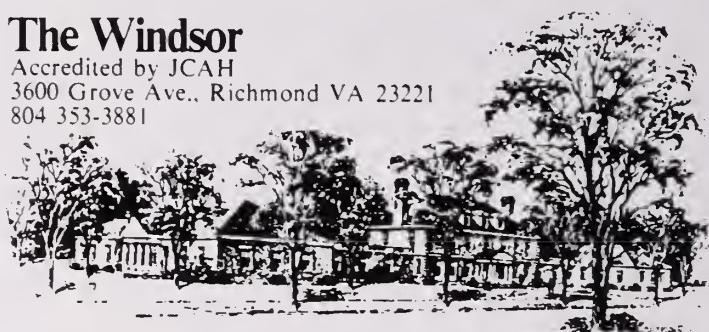
Dr. Pastore was a true Southern gentleman, a very kind and a very gracious person. He endeared the hearts of those who had the privilege to meet him, and it is unlikely he ever met a person he did not like. He was a devoted husband and father with strong religious convictions. He was a friend and a humanitarian par excellence.



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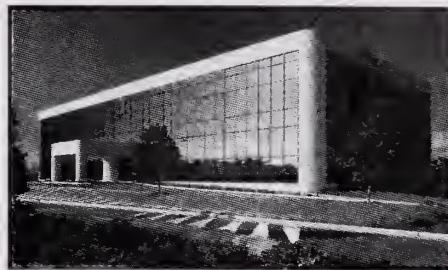
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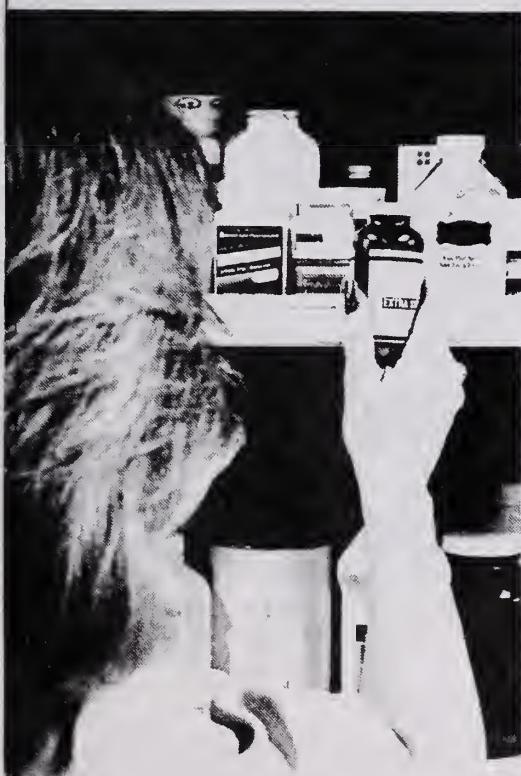
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Diabetes and Vascular Disease (Eastern Virginia Medical School), *Virginia Beach*. CME Office, 804-446-6140.

August 24-27

12th Edition, Practical Dermatology for the Primary Care Physician (Eastern Virginia Medical School), *Orlando, Florida*. CME Office, 804-446-6140.

September 7-9

Annual Meeting/Scientific Session of the Virginia Vascular Society, *The Homestead, Hot Springs*. Dr. Jesse Davidson, program chairman, 703-345-1561.

September 8

Regular meeting of the Medical Society of Virginia's Council, Richmond. James L. Moore, Jr., 804-353-2721.

September 8-11

Addictions Conference (Oxford Institute Network of Care), *Hilton Head Island, South Carolina*. Mary Johnson, 1-800-726-4044.

September 10-14

Basic Course in Nosocomial Infection Control (University of Virginia), *Charlottesville*. Debbie Crickenberger, 804-924-2777.

September 13-14

Pediatrics for the Practitioner: Update 1990 (Johns Hopkins), *Baltimore*. 14.5 credit hrs. Physician fee: \$235. CME Office, 301-955-2959.

September 14-16

Plastic Surgery of the Eyelids and Orbit/Oculoplastic Surgery, a Multidisciplinary Symposium (Eastern Virginia Medical School), *Norfolk*. CME Office, 804-446-6140.

September 27-28

5th Annual Postgraduate Radiology Course: Update in Film Screen Mammography (University of Virginia), Jefferson Sheraton Hotel, *Richmond*. 15 credit hrs. Physician fee: \$300. R.L. Boswell, 804-924-9387.

September 28

13th Annual Perinatal Conference (Medical College of Virginia/VCU), *Richmond*. CME Office, 804-786-0494.

October 3-5

Recent Advances in Clinical Medicine (University of Virginia), Omni Hotel, *Charlottesville*. Lauvonda Young, 804-924-2090.

October 3-6

16th Annual Topics in Gastroenterology and Liver Disease (Johns Hopkins), *Baltimore*. 23.5 credit hrs. Physician fee: \$425. CME Office, 301-955-2959.

October 4-6

Virginia Occupational Health Conference (Medical College of Virginia/VCU), *Richmond*. CME Office, 804-786-0494.

October 8-11

13th Annual Postgraduate Course in Radiology (University of Virginia), Boar's Head Inn, *Charlottesville*. 20 credit hrs. Fee: \$400. Dr. Theodore E. Keats, 804-924-2781.

October 8-12

Basic Course in Nosocomial Infection Control (University of Virginia), *Charlottesville*. Debbie Crickenberger, 804-924-2777.

October 11-13

Management of the Spinal Cord Injured Patient: Pediatric, Aging, and Elderly (Eastern Virginia Medical School), *Virginia Beach*. CME Office, 804-446-6140.

October 22-27

32nd Annual Emil Novak Memorial Course: Gynecology, Gynecological Pathology, Endocrinology, and High-Risk Obstetrics (Johns Hopkins), *Baltimore*. 44.5 credit hrs. Physician fee: \$650. CME Office, 301-955-2959.

November 1-4

Advances and Controversies in Internal Medicine Seminar (Lloyd Noland Hospital), *the Greenbrier, White Sulphur Springs, West Virginia*. CME Office, 205-783-5276.

November 3

Symposium on Current Controversies in Colon and Rectal Cancer (University of North Carolina at Chapel Hill), *Research Triangle Park, North Carolina*. CME Office, 919-962-2118.

November 8-9

15th Annual Symposium for Clinicians on Hospital Infection Control (University of Virginia), Boar's Head Inn, *Charlottesville*. Debbie Crickenberger, 804-924-2777.

November 29-December 1

Advances in Obstetrics and Gynecology (Medical College of Virginia), *Richmond*. CME Office, 804-786-0494.

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1. A. Morales et al., New England Journal of Medicine: 1221, November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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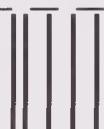
*Division of Survey and Data Resources, American Medical Association, December, 1987

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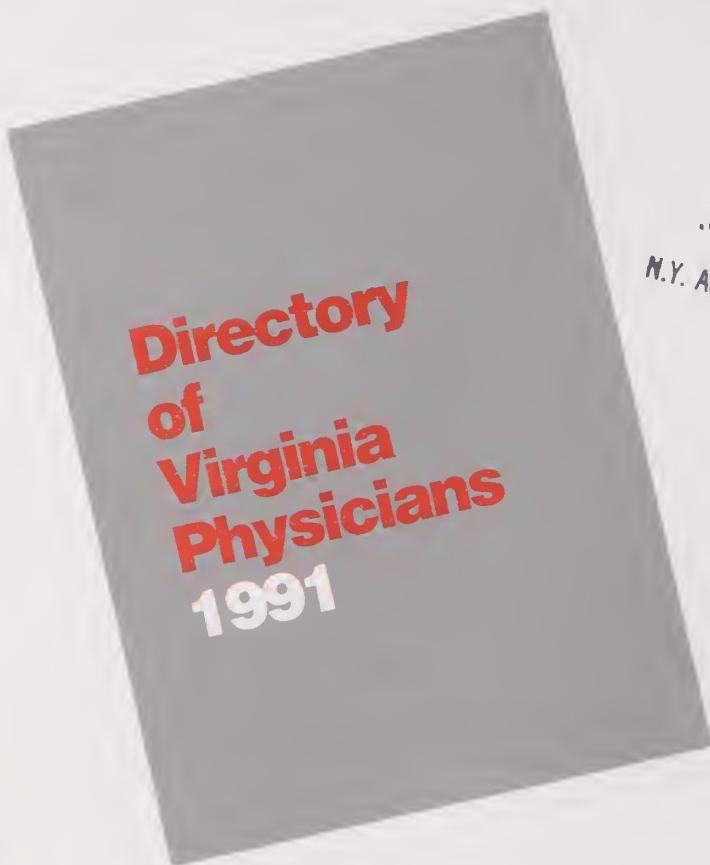
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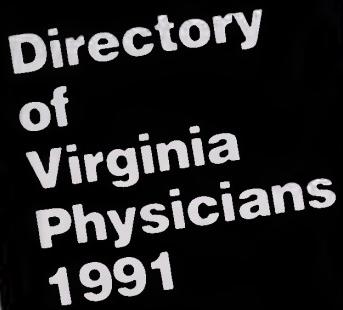
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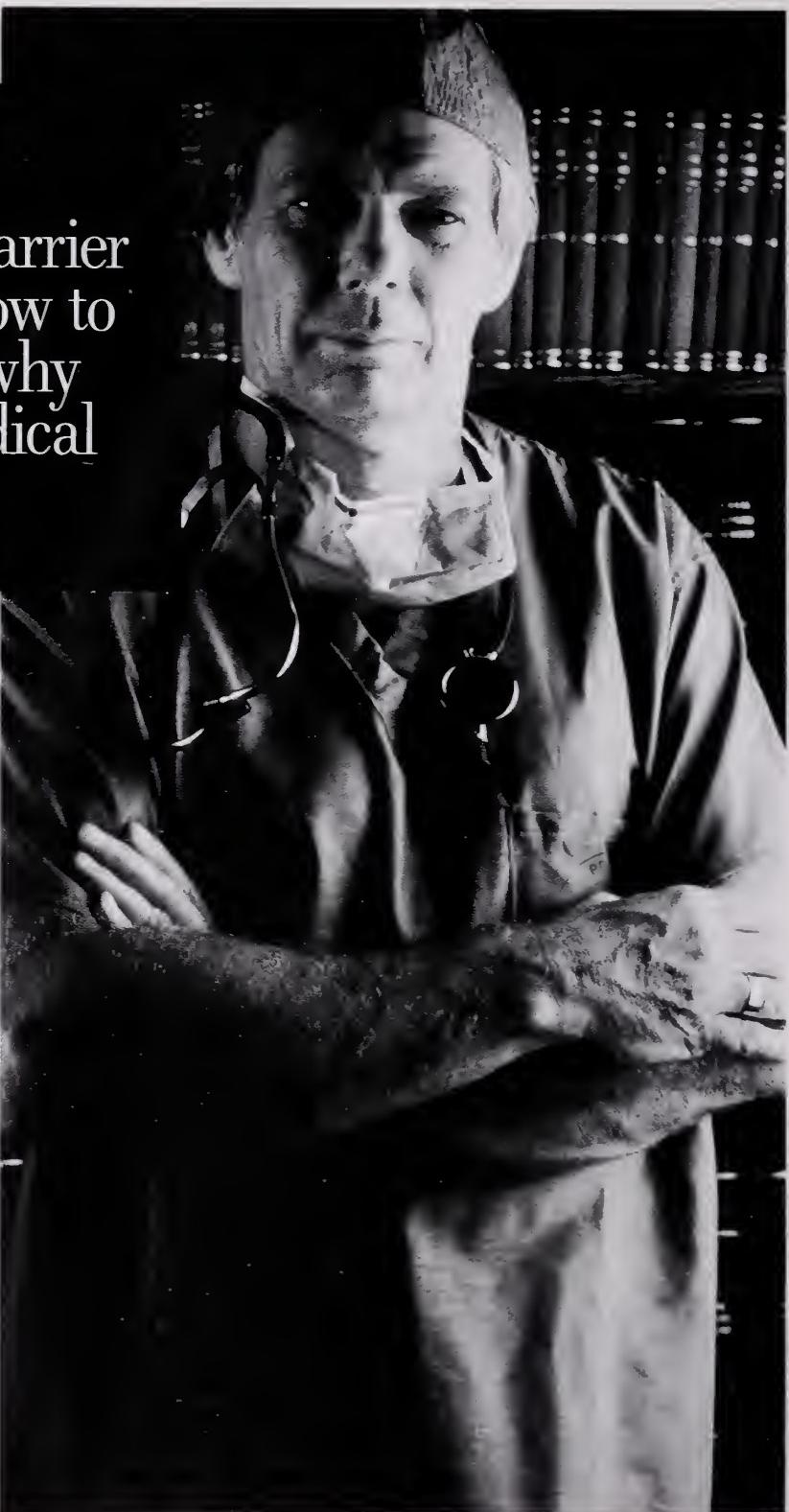
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"A Monstrous Intrusion"**Will National Data Bank Encourage Litigation?**

The National Practitioner Data Bank, which became effective last month, is a monstrous intrusion on the civil liberties of physicians. (Thank heavens they don't have a data bank for lawyers.) On top of the outrageous insult to physicians' privacy,¹ achieved in the name of national peer review, the crazy thing is that the Data Bank may have the opposite effect of what is intended. My associates and I are already beginning to see this in our legal practice. Examples:

1. The Data Bank will discourage settlement of legal claims and encourage malpractice litigation.

While we do very little "insurance" work, we typically represent doctors in malpractice suits as their private counsel. Our job is to ensure that insurance counsel is doing the right things and, most importantly, to act as an advocate for the physician with his or her insurer. In the "old days," in cases with colorable claims we would often, as the doctor's private lawyers, urge an insurance company to settle a case. This would end the doctor's aggravation and protect him or her against possible recoveries in excess of insurance. The hope was we could also protect the doctor's rights against the insurance company if the company acted unreasonably in refusing to settle.

No longer. The Data Bank requires the reporting of any payment of settlement, irrespective of amount. That information is later available to insurers, hospitals, boards of medicine and, under certain conditions, plaintiff's attorneys. Now that the Virginia mal-

practice cap is solidified and assuming that my clients are properly insured to the cap, it would take a severe case indeed for me to urge a settlement. And nuisance settlements would be nuts.

The Data Bank's claim that "a payment in settlement of a medical malpractice action or claim shall not be construed as creating a presumption that medical malpractice has occurred" is . . . well, ludicrous. If settlements don't necessarily mean anything, why is the Data Bank so intent on tracking them down to the last dollar? Is this akin to an F.B.I. "rap" sheet which lists all of a person's arrests and convictions?

The result of all this? Expect fewer settlements of malpractice claims and more litigation, more defense costs, and higher premiums.

2. The National Data Bank will discourage hospital peer review.

A motivating factor behind the Data Bank was to intensify peer review because of the "problem" (I personally don't see a problem) of incompetent doctors moving from one hospital to another and otherwise trying to hide their disastrous practice results from their colleagues. But several of my physician friends with responsible hospital peer review responsibilities have commented about their reluctance to engage in peer review activities which might end up in a doctor's Data Bank file. The perception is that this could "ruin" a physician. As a result, peer review activities will be kept at departmental levels, or be addressed orally or through private understandings. Such lack of formal structure is

what led to the hullabaloo about ineffective peer review not too long ago.

Are my friends right, will a doctor reported to the Data Bank be ruined? Not necessarily, but 1) he or she might expect to deal with the local medical board about the matter (which might require counsel and which is expensive and aggravating); 2) the doctor might have problems with his or her insurance company; 3) he or she might have problems with every hospital in which the doctor has privileges or to which he or she wishes to apply.

3. The National Data Bank will encourage litigation against hospitals over privileges and might lower standards of medical practice.

Doctors who suffer a restriction or diminution of privileges are often required to take some sort of action, if feasible, because of the domino effect of such restrictions upon his or her privileges at hospitals elsewhere.

The Data Bank now also requires that doctors be reported if their request for upgraded privileges is denied (unless that denial relates to failure to meet general standards, like requirement for board certification). A physician who, for example, attends a course, learns a new procedure, duly applies for privileges to do that procedure and is rejected because, say, the department chairman didn't like the course, is reported to the Data Bank. Prior to the Data Bank era I might have advised such a physician to follow appropriate hospital channels and, if unsuccessful, forget about it. Now I'm not so sure. The rejection goes to the Data Bank. If that physician decides to apply for a medical license in the next state, the appropriate board of medicine will learn of the rejection. That will lead to a hearing, most probably, at the respective board's credentials committee, rather than a routine application approval.

What if it leads to a licensure rejection? (The Data Bank keeps track of licensure rejections, too.)

Will hospital doctors, knowing of these potential consequences, grant extensions of privileges that they might not otherwise entertain because of their empathy for the applicant who, if rejected, ends up in the Data Bank? Will this decrease the quality of medicine?

4. Professional societies will shy away from peer review. Many already do because of insurance problems. Now an adverse professional society action which affects membership must be reported to the Data Bank. While such reporting requirements have been in existence with respect to the Virginia Board of Medicine, my belief is that professional societies will be even more reluctant to take—and thus report—peer review type actions because of the intimidating presence of the Data Bank. After all, a physician who is expelled from a professional group for disciplinary reasons will likely sue to reverse the action because of the reporting to the National Data Bank. What professional society can function properly under that type of gun?

The Data Bank is a lawyer's delight. Lucky me. Unlucky us.

—JOHN D. GRAD

John Grad is counsel to the Alexandria Medical Society and a partner in the Alexandria law firm of Grad & Logan.

¹The civil liberties implications of a national computer that keeps tabs on all things wrong, or arguably wrong, "perpetrated" by a select and suspect group of professionals, and which recognizes the "good works" of those who squeal on their colleagues, are obvious. Aside from the faulty underlying premise, the intrusion on privacy and discrimination against doctors as a class, I believe the Data Bank will be counterproductive. That is the thrust of these comments.

Fault/No Fault

Antidote for a Bad System: Rollovers and Easy Offers

You heard what those Yankee doctors in Massachusetts have got going in their legislature? A no-fault medical liability insurance bill.¹ Modeled after workers' comp, funded by 2% surcharge on health insurance policies, makes visions of big cuts in medical liability premiums dance through your head. The Massachusetts Medical Society engineered its introduction early last summer. Late in August I called the Society's headquarters in Boston to see if the bill was still alive.

"Yep, still alive," said the person who came to the phone.

Any bets the bill will pass?

Cheerfully: "Nope. The only bets have been that we wouldn't get it out of committee"—then, elated, "but we did it!" Joint Committee on Insurance was the one reported it out favorably. Next, when the senators come back from recess in mid-September, the Senate Ways and Means Committee. "We're pushing to get it to the floor of the Senate by October."

Amazing. Not limited no-fault, like Virginia's Injured Infant Act, but comprehensive, a whole new system. What, I wondered, does Jeffrey O'Connell think of that? Virginia's Mr. No-Fault.² Saw him not long ago at 4 am on the "Nightwatch" show bulldogging the biggest, orneriest plaintiff's lawyer you ever saw. I rang up the University of Virginia and asked for the John Allan Love Professor of Law. He answered the phone himself.

Yes, he knew of the bill. Innovative. Energetic. Lots of points for Mass Med Soc and its president,

Dr. Barry Manuel, the bill's prime mover. "The present tort system is so bad. These people are trying to apply a more sensible system and that's encouraging."

But do you think it will pass?

Guardedly: "A very, very tough fight." As in all other states, the Massachusetts legislature is loaded with lawyers, many of them plaintiff's lawyers. Plus other special-interest groups circling a bill of this magnitude, waiting to pounce. "Very tough," O'Connell repeated. He sounded dubious.

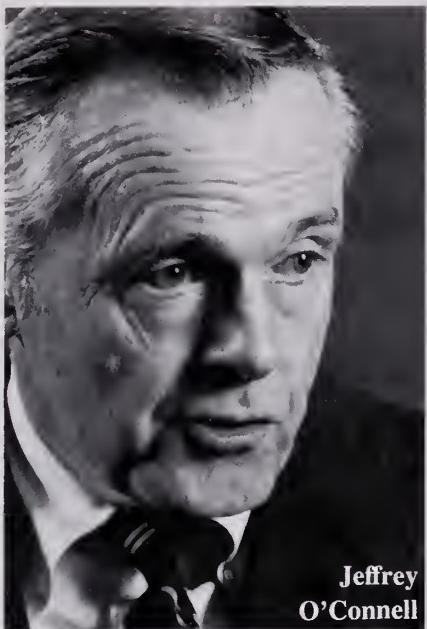
Well, how in the world does anyone get anything past the plaintiff's attorneys?

"You roll over 'em." Then, becoming animated, "You rolled over 'em right here in Virginia with the Injured Infant Act!"

Of course, there were a couple of helpful factors when that was passed, the Professor added. Liability insurance rates had gotten so terribly high, and liability suits had begun popping up in all manner of other professions and businesses and organizations, leading to a hue and cry about astronomical costs and an aroused public. Today, liability rates have subsided somewhat, liability suits are so pervasive they're commonplace, people are worried about other things, that makes it tougher, probably, to get past the plaintiff bar.

You like the Injured Infant Act?

"Well, we don't have any results yet, if that's what you mean, and we won't have results for five or six years more, so what it will achieve remains to be seen, and I will be a little nervous about it until we do. I like what the Act stands for—the



Jeffrey
O'Connell

“You can tinker with the cap or with contingency fees or punitive damages or what have you all you like, but you’ve still got the same bad system.”

first effort to carve out of the tort system a category of injury subject to terribly, terribly high awards. It is a worthy thing to try.”

O’Connell is troubled by the 1990 General Assembly’s revision of the Act’s narrow definition of injury. “It is so hard to decide, in advance, just which victims deserve this limited kind of no-fault compensation and which ones don’t, and then to define the ones who do and the ones who don’t. I’m afraid we will be massaging this definition forever,” he observed.

The 1990 Assembly considered massaging the cap, too, so that it would read \$1 million per defendant, but carried the bill over.³ Forecast, please?

“The cap is perceived as being very hard on the severely injured

people.” But, he pointed out, even if you alter the cap for these people, they still must go through the same long, arduous litigation process with no guarantee of anything in return.

“So you see, these kinds of revisions and extensions only play at the edges of addressing the real problem, which is that the present system is a very bad one and ought to be changed. You can tinker with the cap, with contingency fees or punitive damages or the collateral source rule or what have you all you like, but you’ve still got the same bad system.

“True, the cap that Virginia and other states put on pain and suffering may be one of the factors contributing to the recent stabilization of liability insurance rates, and the fact that rates have stabilized is good—though in point of fact it is relatively insignificant when you consider that they have stabilized at a level that a few years ago would have been intolerable and in many ways is still intolerable. We may have gotten used to them, but that doesn’t lessen the fact that they are still very unmanageable.”

His skepticism about working with the tort system extends to the new arbitration pilot program put together by a coalition of Virginia doctors, trial and defense lawyers, and insurance companies.

“All these things take so much effort,” he said, “but they aren’t really going to make a lot of difference as long as you’ve got the fault system. I would so much rather see all that effort getting behind a different system.”

Like the limited no-fault Injured Infant Act?

“Yes. A new approach.”

Like the no-fault bill in Massachusetts?

“An admirable effort.”

Would you like to see The Medical Society of Virginia sponsor such a bill?

“The passage of the Injured Infant Act encourages me to think that Virginia is more receptive to change than in the past, but even so, I don’t think the time is right for that sweeping a change here,” O’Connell said.

“I would say, Go for the no-fault system, yes, but do it with a long view, 10 or 15 years, take it in small pieces, like the Injured Infant Act, like the no-fault immunization measure. We get reforms every once in a while.”

And he just happened to have at the ready an idea for a small piece of reform he thinks has terrific possibilities. He calls it “Easy Offers,”⁴ he describes it as a halfway house between the tort system and no-fault, and this, in brief, is the way it plays:

Within a certain period of time after a claim is filed, 90 days, maybe, or 180 days, the carrier offers to pay for all economic losses the patient alleges (including medical expenses). Thereupon one of two things follows. 1) Patient accepts, case is foreclosed, that’s that. 2) Patient believes there’s proof beyond reasonable doubt that provider acted “wantonly” (criminally), refuses offer, goes to court for high-dollar punitive damages. This scenario could apply broadly, to all claims, or narrowly, to one category of claim.

Would you recommend Easy Offers legislation as a next step in Virginia?

“I certainly would. Obstetrical cases would be the logical place to start. That’s where the crisis is.”

—ANN GRAY

1. Larkin H. Massachusetts no-fault bill seeks system like workers’ comp. Am Med News June 22, 1990, p 9
2. O’Connell J. The “neo no-fault” alternative. Va Med 1985;112:239-43
3. MSV Legislative Update 1990;3(3):4
4. O’Connell J. Pragmatic constraints on market approaches: a response to Professor Epstein. Va Law Rev 1988;74:1475-83

Hewing to the CPT-4

How to Avoid a Medicare Postpayment Audit

A number of Virginia physicians have been jolted recently by audits conducted by the Travelers Insurance Company advising that Medicare disagrees with their selection of procedure codes for office and hospital visits. Physicians are being notified by an "educational" letter from the Medicare postpayment audit unit.

A typical notification from the Travelers advises that an audit of the physician's claims, payment history, and records for the past one or two years has been conducted by Medicare. The letter explains Medicare's evaluation of the physician's billing practices and requests that "such billing practices cease." The notice concludes with the admonition that continued abuse could result in exclusion from the Medicare program in ac-

cordance with Section 1862(d)(1) of the Social Security Act.

The abusive billing practice referred to is single-level procedure coding. Medicare requires that physician billing for consultations and for office, hospital and home visits conform to the American Medical Association Physicians' Current Procedural Terminology, Fourth Edition, 1990 (CPT-4). That coding scheme provides for multiple levels of service in the office, hospital and home. The levels range from minimal (e.g., blood pressure check by physician employee) to comprehensive (complete indepth evaluation and examination of the patient). CPT-4 provides five levels of service for initial consultations, new patient office visits, hospital care (subsequent to admission) and established

patient home visits. Six levels of care are available for established patient office services.

Medicare audits are focusing on physicians who bill all or most visits at only one level of service. Medicare expects that a typical physician practice would include some patients requiring service only at the minimal or brief level; some needing intermediate, extended or comprehensive service; but the majority requiring limited levels of service. Physicians who, for example, routinely bill all hospital visits at the 90260 (intermediate) level create a billing pattern that appears aberrant to the Medicare postpayment analysis computer program. These physicians are selected for indepth audits which can result in issuance of the "educational" missive.

Although the first letter to a physician is indeed only educational, it certainly implies more drastic action in the future if the physician doesn't react to Medicare's suggestions. Medicare has at its disposal a variety of punitive measures, up to and including exclusion from the Medicare program. However, the usual Medicare procedure in this case is to conduct a followup audit within 6-18 months after the initial educational letter. If the billing pattern remains unaltered, Medicare reviews a random sample of physician records to determine how many services may have been "up-coded." Extrapolating this sample to the universe of claims for the period reviewed, Medicare calculates an overpayment and notifies the physician that he has 30 days to repay the entire amount. Such

MEDICARE CODING GUIDE	Minimal	Brief	Limited	Intermediate	Extended	Comprehensive
Services Performed	1 of 2	3 of 4	4 of 6	5 of 7	8 of 11	10 of 13
Complaint/Symptoms						
Interval History		<input type="checkbox"/>				
Details of Illness						
Review of Systems						
Past History	<input type="checkbox"/>					
Family History						
Blood Pressure						
Temperature						
Pulse						
Limited Exam (One area)		<input type="checkbox"/>				
Limited Exam (Two or more areas)			<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Complete Exam (Including pelvic)				<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Lab/X-ray Review		<input type="checkbox"/>				
Diagnosis/Problem						
Advice/Treatment/Injection	<input type="checkbox"/>					
Office: New Patient	90030	90000 90040	90010 90050	90015 90060	90017 90070	90020 90080
Established Patient						
Hospital:		90240	90250	90260	90270	90280
New Patient	90130	90100 90140	90110 90150	90115 90160	90117 90170	
Established Patient						

New: Physician's Guide to Durable Power of Attorney for Health Care

overpayments for a year's worth of claims can easily run into many thousands of dollars.

Happily, physicians can easily avoid this somewhat unpleasant encounter with Medicare's enforcement unit. The most important first step is to become familiar with the Definitions and Items of Commonality section in the CPT-4 Introduction (pp 19-26). In conducting its audits of patient charts and hospital records, Medicare looks for documentation that the level of service billed is consistent with the code described in CPT-4.

Following a patient encounter, the physician should indicate on the patient chart (or checkoff form) the level of service code commensurate with the service actually performed. In some practices, this responsibility is delegated to a coding or billing specialist. That person should be familiar with the CPT-4 definitions and the necessity to utilize all levels of service when appropriate.

Physicians may also want to utilize the accompanying chart as a quick reference source when selecting the appropriate coding level.

DAVID J. LATHAM, JD

Attorney Latham is associated with the law firm of Grad & Logan, PC, Alexandria, concentrating on health-care reimbursement and medical/legal issues. Address correspondence to him at 9702 Gayton Road, # 220, Richmond VA 23233.

Submitted 6-4-90.

A second legal instrument in the surrogate decision-making category has been enabled by Virginia's General Assembly (Va Code §37.1-134). It is the "durable power of attorney for health care," a variation on the classic legal document for financial affairs. This new instrument differs from the familiar Natural Death Act declaration in a highly significant way: It is not limited to terminal situations.

There are other important differences as well, and the Editors commissioned the Medical Society of Virginia's counsel, Sandra L. Kramer, to develop a *Physician's Guide to Durable Power of Attorney for Health Care*. This guide tells you exactly what to look for and how to handle this new document. With it you will receive a sample form for executing a durable power of attorney for health care. *Guide* and form are offered to you here free of charge.

Many attorneys suggest

executing both a durable power of attorney for health care and the Natural Death Act declaration form, and some of your patients may do so. Requests for the latter form, which Va Med has been sending out since 1983, took a terrific leap upward after the U.S. Supreme Court's decision in the Cruzan case. There are plenty of copies on hand for your patients, and copies are available as well of the companion piece, the *Physician's Guide to the Natural Death Act*. Both are free of charge.

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Patient Access to Medical Records: Answers to Ten Common Questions

1 What exactly do "medical records" include? Should I include billing records? How about medical records prepared by other physicians or health facilities?

The Virginia Public Records Act defines "medical records" as "the documentation of health care services, whether physical or mental, rendered by direct or indirect patient-provider interaction, which is used as a mechanism for tracking the patient's health care status." This Act applies to records in the custody of public agencies and is not directly applicable to records held by private physicians, but it gives some indication of the scope of the term. Billing records would not appear to fall within this definition, but medical records prepared by another physician might well be included.

2 What rights does a patient have to his or her medical records?

Virginia law provides that upon written request, patients are entitled to copies of their medical records within 15 days at a reasonable charge. This right is enforceable by a subpoena issued by the court.

3 Can I refuse to give a patient with an outstanding bill access to his or her records?

No. Virginia law provides that a patient must be furnished with copies of his or her medical records on request and does not provide an exception for patients with outstanding bills.

4 Are there any limitations on patients' access to their medical records?

Virginia law provides that mental records which are official records may not be reviewed by a patient if the treating physician has made a part of these records a written statement of his opinion that a review of such records by the patient would be injurious to the patient's physical or mental well-being. The same pertains when a patient requests copies of mental rec-

ords, although in the case of copies, the physician may be required to give the records to the patient's attorney.

5 Can I bill for the copying costs? If so, is there a specified charge?

Yes. As currently written, Virginia law provides that "a reasonable charge, not to exceed 50¢ per page," may be made for such copies. However, on the initiative of the State Legislative Committee, the Medical Society of Virginia intends to ask the 1991 General Assembly to rewrite the passage so that not only photocopying but associated costs, such as research, are chargeable.

6 Can I refuse a minor patient's parents, who pay for the treatment, access to the medical records of that patient?

Virginia law does not address this issue directly. However, minors are deemed adults for purposes of consenting to medical services in connection with 1) venereal or infectious diseases which must be reported to the State Board of Health; 2) birth control, pregnancy, or family planning; 3) mental illness; and 4) substance abuse. Physicians may have a duty of confidentiality to minor patients who can give consent to treatment. If so, the relevant records should not be released without the authorization of the minor's parents.

7 What if I no longer have the requested records?

You should so inform the patient, and if you know where the records may be found, you may want to tell the patient. Virginia law provides that a patient may seek a subpoena to force a physician to deliver copies of medical records and provides that a court may impose damages, including costs and attorney's fees, upon a physician who willfully or arbitrarily refuses to provide them. If you have exerted your best efforts and cannot obtain the rec-

ords, then you should not be penalized.

8 Should I transfer original medical records?

Virginia law does not address this issue. The common law indicates that original medical records are the property of the physician who created and maintains them, but a patient has an interest in the information in the records. The prudent course would be to retain the original records and transfer copies.

9 Who can sign an authorization for records release?

Virginia law does not address this issue. The common law indicates that a patient may authorize the release of his or her records or, if the patient is incompetent, his or her guardian may do so.

10 Who can get access to a deceased, incapacitated, or incompetent patient's records?

These records should be provided on request to the following persons in the following order of priority: 1) the personal representative of a deceased patient; 2) the legal guardian or committee of an incompetent or incapacitated patient; 3) the spouse; 4) an adult son or daughter; 5) either parent; 6) an adult brother or sister; 7) any of the other relatives of the patient in the descending order of blood relationship. Physicians are required to provide these records only if they are not aware of any available person with a higher priority.

—TIMOTHY A. HARTIN

Some of the answers above are based on Virginia statutes; others are interpretations of the case law in other jurisdictions. Physicians are encouraged to contact their attorneys for more specific guidance.

SOMETHING TO THINK ABOUT...

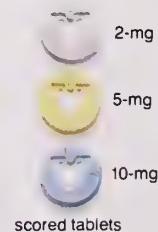
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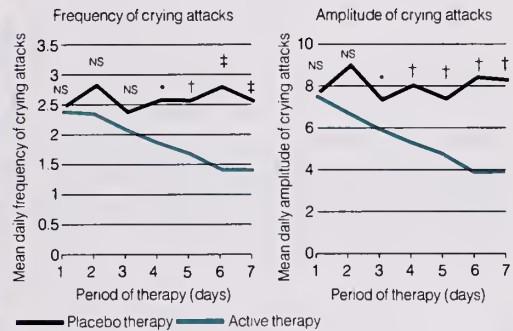
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1. Kanwaljit SS, Jasbir KS. Simethicone in the management of infant colic. *Practitioner*. 1988;232:508.

Liver Transplantation in Virginia

Mitchell L. Shiffman, MD, Marc P. Posner, MD, Velimir Luketic, MD,
A. Scott Mills, MD, Wallace Berman, MD, Ann Whitman, RN,
H. M. Lee, MD, and Robert L. Carithers, Jr., MD, *Richmond, Virginia*

Survival rates ranging from 70-80% have been documented over the past three years in liver transplant patients at the Medical College of Virginia. Survival declined in patients with numerous complications of chronic liver disease and with significant deterioration in hepatic synthetic function. Early referral was a major factor contributing to survival.

LIVER transplantation is now well accepted as an effective long-term treatment for patients with end-stage liver disease. In Virginia, liver transplantation began in earnest at the Medical College of Virginia (MCV) in January 1984. Since then over 100 transplants have been performed in 82 adults and 13 children. Overall, one-year patient survival during the past three years has ranged between 70-80%, equivalent to that reported by other centers.^{1,2}

Four years ago we reviewed the history of liver transplantation and the early results of our program for VIRGINIA MEDICAL.³ The present article is designed for the practitioner and specialist caring for patients with liver disease in Virginia and the surrounding region. Key issues regarding referral of patients for liver transplant evaluation are discussed as are some

From the Division of Gastroenterology, Section of Hepatology (Drs. Shiffman and Luketic), the Division of Vascular and Transplant Surgery (Drs. Posner and Lee and Nurse Whitman), and the Departments of Pathology (Dr. Mills) and Pediatrics (Dr. Berman), Medical College of Virginia/Virginia Commonwealth University. Dr. Carithers is now with the University of Washington School of Medicine at Seattle. Address correspondence to Dr. Shiffman at Box 711, MCV Station, Richmond, VA 23298.

Submitted 3-22-90.

of the long-term complications associated with hepatic transplantation. These issues are of major concern to referring physicians who share the burden for the future care of these patients.

The Team

The complex and multiple issues and disciplines required to adequately care for patients with chronic liver disease in need of hepatic transplantation demands a team approach. This philosophy is shared by many other transplant centers and the essential personnel for such an endeavor was discussed in a recent review.⁴ At MCV the liver transplant program is guided by a multidisciplinary committee which meets on a regular basis to discuss the pre-, intra-, peri-, and post-transplant needs of each patient. This committee is composed of key personnel from the transplant team, which includes not only transplant surgeons and dedicated anesthesiologists but also transplant hepatologists, critical care/pulmonary specialists, a liver pathologist, blood banking specialists, a social worker, psychologist, a hospital clergy representative and the liver transplant nurse coordinator. The roles that many of these specialists play in the transplant process are discussed below.

At MCV, as in most other successful programs, the

transplant hepatologist serves as the referral agent for the practicing physician and directs the pretransplant evaluation to determine if the patient is a suitable candidate for transplantation. Since most patients do not require emergent transplantation after their evaluation the hepatologist also works closely with primary referring physicians to manage the complex medical care that patients with chronic liver disease require prior to their transplant. During this waiting period, which may last anywhere from days to months, the progress of each patient is closely followed by the interdisciplinary transplant committee.

The transplant surgeon is the leader of the surgical team and is notified whenever a potential donor organ becomes available. He must then decide which patient on the active waiting list is the most appropriate candidate to receive this organ. Four major criterion are utilized to match a donor liver to a potential recipient: 1) ABO blood group compatibility, 2) body size of donor and recipient, 3) the recipient's overall clinical condition, and 4) the quality of the donor organ. The blood bank is then contacted to ensure that an adequate supply of blood products is available to support the operation, and arrangements to retrieve the organ and have it transported to MCV are made. In most cases this requires the surgeon to travel long distances by corporate jet, harvest the organ and then return to MCV prior to starting the transplant operation. However, if an experienced transplant surgeon is on staff at the donor hospital, arrangements can be made for this surgeon to remove the organ and have it sent to MCV on ice. Such arrangements are becoming increasingly common throughout the country as the number of trained transplant surgeons increases. In addition, the development of a new preservative (University of Wisconsin solution) has increased the amount of time a liver can be stored to nearly 24 hours,⁵ enabling the surgical team time to rest and refresh themselves between harvesting and transplanting operations.

The transplant operation itself is a tedious and complex process taking from 6-18 hours and requiring large amounts of blood products. We and others have described this procedure in recent reviews.^{3,6,7}

Following the transplant operation the patient is moved to the medical intensive care unit and cared for by a team of intensive care unit/pulmonary specialists and the transplant hepatologist, all under the overall direction of the transplant surgeon. The patient remains in this unit until clinically stable, typically 5-10 days, before being transferred to the Clinical Transplant Center. The patient remains in this specialized nursing unit anywhere from 2-4 weeks, until ready for hospital discharge.

Following hospital discharge the hepatologist and liver transplant nurse coordinator assist referring physicians with post-transplant followup, and regularly monitor blood cyclosporin levels and liver function

tests. Liver biopsies are performed at regular intervals and/or whenever necessary to investigate fluctuations in serum tests of liver function.

A highly skilled pathologist interested and experienced in liver disease is an essential member of any successful liver transplant program. This individual interprets pre-transplant liver biopsies, evaluates the explanted recipient liver for signs of occult carcinoma, and interprets all biopsies from the transplanted allograft. Although hepatic allograft rejection is frequently associated with elevations in serum liver transaminases (AST and ALT) this diagnosis can only be confirmed by histologic examination of the liver. Since this histologic picture is often quite subtle it requires both skill and experience to be confident of this diagnosis. This distinction is critical since rejection is treated with large doses of potentially toxic immunosuppressive agents.

The clinical nurse coordinator is the primary contact person for patients and their families, with input into nearly every aspect of patient care. The nurse schedules the initial transplant evaluation, orients patients and their families to the transplant center, and educates patients about such issues as the transplant process, immunosuppression, side-effects of drugs, and post-operative complications. Following transplantation the nurse coordinator monitors scheduled blood tests and cyclosporine levels, reviews these with transplant physicians, and schedules any necessary tests or biopsies to investigate laboratory abnormalities. Without a doubt, an effective nurse coordinator may be the most important asset to any transplant program.

Indications/Contraindications

Indications for liver transplantation include chronic or acute liver failure of any etiology. A list of the most frequent diagnoses for patients undergoing liver transplantation throughout the country are listed in Table 1. In addition, several genetic diseases which manifest their deleterious effects via the liver can be cured by hepatic transplantation. Such diseases include homozygous type II hyperlipidemia, hemophilia (homozygous factor VIII deficiency), alpha-1-anti-trypsin deficiency, glycogen storage diseases, and certain types of porphyria, to name a few. Table 2 lists the percentage of liver transplants performed at MCV during the past six years according to liver disease etiology. Special considerations for several of these are discussed below.

Only five absolute contraindications to liver transplantation currently exist: 1) acquired immunodeficiency syndrome (AIDS); 2) active sepsis outside the hepatobiliary tract; 3) cholangiolar carcinoma; 4) advanced cardiopulmonary disease; and 5) metastatic cancer.^{1,3} Chronological age is no longer an important factor as long as the patient has no significant underlying cardiopulmonary complications. The oldest pa-

tient transplanted at MCV is now 66 years of age and continues to do well three years after his operation.

Alcoholic Liver Disease. Clearly, the most debatable indication for liver transplantation is alcoholic liver disease. Many transplant centers require that

patients be abstinent for at least six months prior to undergoing transplantation. However, this approach has been challenged by Starzl and his colleagues from Pittsburgh, who reported that patients with alcoholic liver disease had a one-year survival of 73%, no different than that observed for other liver diseases.⁸ Only one of these patients returned to alcoholism despite the fact that none were given any formal counselling or treatment for their disease.

At MCV 7% of our patients have undergone transplantation for alcoholic liver disease. To our knowledge none has returned to drinking alcohol. We do not discourage the referral of patients with alcoholic liver disease for transplantation and assess each patient on an individual basis. However, we do insist that patients are abstinent for at least six months, and we strongly encourage them to participate in a formal counselling program prior to transplantation.

Our major concern about transplanting patients with alcoholic liver disease is the presence of underlying but clinically undetectable extrahepatic manifestations of chronic alcoholism, such as cardiomyopathy and neuropathy. Indeed, 50% of our patients transplanted for alcoholic cirrhosis have sustained an episode of acute cardiac decompensation anywhere from three days to 12 weeks following their transplant despite a normal pre-operative cardiac evaluation.

Hepatic Malignancies. The risk of hepatocellular carcinoma (HCC) in patients with underlying cirrhosis is significantly higher than that observed in the general population. As a result, it is not unusual to find a small incidental HCC after a cirrhotic liver has been removed and thoroughly examined. In the Pittsburgh experience 14 patients had tumors found in such a manner. Of these, only one had recurrent disease after 3-73 months of followup.⁹ At MCV, such "incidental" hepatomas have been found in two patients (2.5%) following removal and careful inspection of their cirrhotic liver. Both are still alive without evidence of metastatic disease up to three years following transplant.

In contrast to the incidental tumor, liver transplantation specifically for known unresectable HCC has produced disappointing results. Ninety percent of patients had recurrent tumor within two years of transplantation.⁹ At MCV we have performed one transplant for unresectable hepatoma. Despite a pre-transplant laparotomy, no sign of metastatic disease at the time of transplantation, and histologic evidence that the tumor was confined within the capsule when removed, recurrent HCC appeared within one year. Thus we are reluctant to perform transplantation for unresectable primary liver cancer, especially with the scarcity of donor organs.

Cholangiocarcinoma is a particularly distressing problem. Not only can this tumor masquerade as sclerosing cholangitis, but 12-15% of patients transplanted for well-documented sclerosing cholangitis are

Table 1. Common Indications for Liver Transplantation.

Chronic hepatocellular liver diseases
Chronic viral hepatitis (B, C and B + D)
Autoimmune chronic active hepatitis
Alcoholic liver disease
α-1-antitrypsin deficiency
Congenital hepatic fibrosis
Cryptogenic cirrhosis
Hemochromatosis (unresponsive to phlebotomy)
Wilson's disease (unresponsive to penicillamine)
Chronic cholestatic liver diseases
Primary biliary cirrhosis
Sclerosing cholangitis
Secondary biliary cirrhosis
Caroli's syndrome
Biliary Atresia
Vascular diseases
Budd-Chiari syndrome
Veno-occlusive disease
Fulminant Liver Failure
Viral hepatitis (B, C, B + D)
Wilson's disease
Drug or toxin induced
Inherited Metabolic Diseases
Hemophilia A
Homozygous familial hyperlipidemia
Tyrosinemia
Glycogen storage diseases
Protoporphyria

Table 2. Etiology of Liver Disease in 136 Adults and 23 Children Evaluated January 1984-February 1990.

	% of Total Pts Evaluated	% of Total Pts Transplanted*
Adults		
Chronic active hepatitis	21%	18%
Primary biliary cirrhosis	17%	18%
Sclerosing cholangitis	12%	16%
Chronic hepatitis B	11%	11%
Cryptogenic cirrhosis	11%	10%
Alcoholic cirrhosis	5%	7%
Post-transfusion chronic non-A, non-B hepatitis (probably hepatitis C)	5%	5%
α-1-antitrypsin deficiency	4%	3%
Fulminant hepatic failure	4%	3%
Primary graft failure	2.5%	3%
Hemochromatosis	2.5%	1%
Cholangiocarcinoma	1.5%	2%
Tylenol overdose	1.5%	0%
Hepatocellular carcinoma	<1%	1%
Chronic hepatitis D	<1%	1%
Bile duct breakdown		
Requiring retransplantation	<1%	1%
Children		
Biliary atresia	61%	69%
α-1-antitrypsin deficiency	9%	15%
Neonatal hepatitis	9%	8%
Choledochal cyst	4%	0%
Tyrosinemia	4%	0%
Chronic active hepatitis	4%	8%
CMV hepatitis	4%	0%

* Total number of patients transplanted: 82 adults, 13 children

found to have cholangiocarcinoma after their liver is removed and carefully inspected.⁹ Unfortunately, the prognosis for each is poor. All patients with any evidence of cholangiocarcinoma at transplantation typically develop metastatic disease and do not survive beyond two years. Our experience is similar to that reported by other transplant centers. Of patients transplanted at MCV for presumed sclerosing cholangitis 2/12 (14%) were found to have had cholangiocarcinoma throughout the liver. An additional 7% of patients were found to have incidental cholangiocarcinoma in association with sclerosing cholangitis. All but one of these patients subsequently died or developed metastatic disease within one year of transplant. Our current feeling is that patients with well-documented sclerosing cholangitis should be transplanted early in their course and that the entire recipient common bile duct be resected.

Chronic Hepatitis B Infection. All patients transplanted for chronic hepatitis B virus (HBV) infection develop reinfection of their donor organ and remain chronic carriers of HBV. Treatment with large quantities of hepatitis B immune globulin and/or immunization against HBV immediately prior to and at the time of transplantation does not prevent reinfection.⁶ However, the clinical course of these patients remains highly variable. Although some develop aggressive chronic active hepatitis and cirrhosis, many simply remain asymptomatic carriers of HBV. Of the patients with chronic HBV infection transplanted at MCV, 50% have developed cirrhosis. Despite this, we continue to evaluate and transplant persons with cirrhosis and chronic HBV infection but fully explain the risk of recurrent disease. The use of new treatments for HBV infection, such as alpha-interferon administered weeks to months prior to transplantation, may reduce the rate of recurrent HBV infection in the future.

Fulminant Hepatic Failure. Transplantation for patients with fulminant hepatitis is often a "catch-22." Spontaneous survival rates for patients with stage I or II encephalopathy are good. Transplantation at these stages is rarely necessary and would leave a foreign organ in a patient who would have otherwise recovered. In contrast, if one waits until the patient is in stage IV coma, where survival is less than 10%, brain death is common, despite successful transplantation. The timing for transplantation is even more difficult since the rate of deterioration from stage II-IV encephalopathy may occur within hours. As a result, a donor organ is often not available in time to save the patient. Over the past four years eight patients with fulminant hepatic failure have been evaluated for emergent transplantation at MCV. Four recovered spontaneously, two reached stage IV coma and died before a donor organ could be obtained, and two patients underwent transplantation. Unfortunately, the timing was not adequate and both suffered irreversible neurologic damage in spite of well-functioning allografts.

Thus we and others¹⁰ feel that the best results for patients with fulminant hepatic failure will be achieved only by early referral to a transplant center for continuous neurologic monitoring with transplantation at the first sign of deterioration.

Referral of Patients

Early referral of patients with known chronic liver disease for transplant evaluation is one of the most important predictors for a successful outcome. Waiting until patients with chronic liver disease develop signs of hepatic decompensation severely restricts their ability to survive the waiting period and surgical procedure. In addition, patients referred late in their disease are also at increased risk for post-operative complications and have a longer post-operative hospitalization. Such complications significantly increase medical costs and decrease patient survival. Two large series have clearly documented that survival approaches 90% when patients are referred for transplant evaluation early in their course and transplanted when relatively healthy, rather than after complications have occurred and patients begin to deteriorate.^{1,2} In contrast, transplantation on an emergent basis reduces survival to less than 25%.

These same trends have been observed in our program. Long-term survivors of liver transplantation at MCV had slightly better overall hepatic function at the time of evaluation as determined by serum bilirubin, albumin and pro-time compared to persons who died (Table 3). Of critical importance, however, was that

Table 3. Clinical and Laboratory Features of Patients at Time of Liver Transplant Evaluation and Outcomes, January 1984-February 1990.

	Long-Term Survival	Died Post-Transplant	Died Pre-Transplant
Number of Patients	58	24	25
Clinical Features			
Variceal bleeding	26%	14%	76%*†
Ascites	74%	56%	68%
SBP	22%	14%	50%*†
Hepatic encephalopathy	32%	46%	76%
Laboratory Values			
Bilirubin (mg/dl)	9.4 ± 0.5	16.0 ± 3.1	18.3 ± 3.1*
Albumin (mg/dl)	3.1 ± 0.1	3.0 ± 0.1	2.9 ± 0.1
PT (seconds)	14.8 ± 0.2	16.1 ± 0.8	18.4 ± 0.9*†

Values are given as mean ± SE.

SBP: spontaneous bacterial peritonitis.

PT: Prothrombin time.

* Significantly different than for long-term survivors; p < .05.

† Significantly different than for patients who died following transplantation; p < .05

patients who died before a donor organ could be found had significant increases in serum bilirubin and prothrombin time, a lower plasma albumin, and significant increases in the number of major complications associated with chronic liver disease, i.e., variceal bleeding, ascites and spontaneous bacterial peritonitis (SBP), compared to patients who survived long enough to undergo transplantation, regardless of out-

come (Table 3). This suggests that these patients were referred for transplantation far too late in their course and had little hepatic reserve to survive the evaluation and waiting process.

It is therefore apparent that patients with chronic or fulminant hepatic disease should be referred for liver transplant evaluation at the first sign of hepatic compromise, if not sooner. A list of the symptoms we consider to be ominous signs of progressive liver disease and which should prompt evaluation are listed in Table 4. Even the slightest abnormality in hepatic

Table 4. Indications for Liver Transplant Evaluation.

Chronic Liver Disease
First appearance of ascites
First episode of encephalopathy
First variceal bleed
Hyponatremia (serum sodium <130 mM)
Any evidence of hepatic synthetic dysfunction
Hypoalbuminemia (albumin <normal)
Prothrombin time ≥2 seconds than control
Fulminant Liver Failure
First appearance of encephalopathy
Prothrombin time ≥4 seconds over control without correction
Bilirubin >10 mg/dl
Serum creatinine >2.0
Serum albumin <3.5

synthetic dysfunction in patients with known chronic liver disease is important. Subtle declines in serum albumin and the development of a mild coagulopathy are often the first signs of hepatic decompensation. However, these signs are frequently overlooked until more dramatic complications appear. Indeed, the higher incidence of bleeding, ascites and SBP observed in patients who survived transplantation compared to patients who died following this procedure reflects their referral earlier in the course of their disease. Although patients who died following transplantation had less complications, their hepatic function (based upon measurements of bilirubin, albumin and prothrombin time) was more severe (Table 3). Since the natural course of end-stage liver disease is highly variable and unpredictable, the benefits of early referral for transplant evaluation are therefore apparent.

The evaluation procedure itself is fairly straightforward and is designed to answer four basic questions: 1) Can the patient tolerate a transplant operation? 2) What does the hepatic vasculature look like? 3) Has a treatable cause of this patient's liver disease been overlooked? 4) Is the patient psychologically, emotionally and financially able to undergo this procedure? The patient is evaluated for underlying cardiac, pulmonary and renal disease. A hepatic angiogram is performed to evaluate the hepatic artery and portal vein for anomalies and patency. If not done so previously, all screening tests for treatable causes of chronic active hepatitis are performed as well as a liver biopsy if not contraindicated by the presence of a

coagulopathy and/or thrombocytopenia. The complete list of tests included in our pre-transplant evaluation are listed in Table 5.

Table 5. Tests in Liver Transplant Evaluation.

Etiology of Liver Disease: ceruloplasmin, ferritin, transferrin, AMA, anti-smooth muscle antibody; anti-mitochondrial antibody, α-1-antitrypsin level, serologies for Hepatitis B (sAgm sAb, cAb), C (Ab), D (Ab if pt HBV+); liver biopsy (evaluation of any previous biopsies; if previous biopsy not available and not contraindicated by coagulopathy and/or thrombocytopenia).
Cardiovascular System: persantine or stress-thallium test; cardiac catheterization (if P-thallium test + or age >55 yrs); ECHO cardiogram; EKG.
Pulmonary System: pulmonary function tests; arterial blood gas.
Hepatic Blood Supply: ultrasound with duplex scanning; mesenteric angiogram.
Renal Function: Serum BUN and creatinine; 24-hr urine for creatinine clearance.
Psychosocial: psychological evaluation, evaluation of health insurance coverage and copayments.

With early referral, many patients with chronic liver disease will not be either medically or physically ready for transplantation following their evaluation. This is actually the optimal situation. Once the evaluation is complete patients can be comfortably but closely followed by their referring physician with periodic input from the transplant hepatologist. The patient can then be moved to the active transplant waiting list at the first hint of further decline.

Early referral for transplant evaluation also gives the patient time to adapt psychologically to the realization of this procedure, to tour our facilities, and to meet other patients who have undergone hepatic transplantation through our program. It is equally important that patients have time to assess the financial impact that this procedure might have on their family and/or to raise money in order to afford this operation. This later point is particularly important and is discussed further below.

Long-Term Complications

Of the long-term complications of liver transplantation discussed in our recent review,¹¹ we discuss here three major long-term concerns to patients and referring physicians: rejection of the donor organ, recurrence of chronic liver disease, and the risks of life-long immunosuppression.

Allograft Rejection. Approximately 75% of patients will experience a mild rejection episode within 2-4 weeks of transplant. Although this is suggested by elevations in bilirubin, AST and ALT, and an abrupt decline in bile production,¹² allograft rejection can only be diagnosed by liver biopsy. The histologic features of rejection include portal inflammation with a mixed mononuclear/polymorphonuclear leukocyte infiltrate, erosion and destruction of small bile ducts and lymphocytic invasion of portal and central venous endothelium.¹¹

In the outpatient setting acute rejection should be suspected in patients with flu-like symptoms, fever, malaise, and mild increases in AST, ALT and bilirubin. Most liver transplant recipients have a normal liver profile. Thus any elevation in blood chemistries may be significant. However, since both the clinical and laboratory features of rejection are non-specific and treatment with high dose immunosuppressive medication is not entirely benign, a liver biopsy must be performed prior to treatment in every case. Therefore all patients with suspected rejection should return to the transplant center for liver biopsy and treatment if necessary.

Chronic rejection is characterized by bile duct drop-out and is frequently referred to as the "vanishing bile duct syndrome." This condition is typically preceded by an episode of acute rejection which never fully responds to treatment. It is associated with chronic elevations in serum AST, ALT and bilirubin, is progressive, irreversible and eventually requires retransplantation.¹¹ Although the incidence of chronic rejection is reported to be 10%, we have not seen this in any of our patients treated with our current triple drug immunosuppressive regimen (see below).

Recurrence of Chronic Liver Disease. As noted above, all patients with chronic hepatitis B virus develop recurrent infection in the new liver. The majority of patients progress to cirrhosis, although the clinical course of post-transplant hepatitis B is highly variable.

Recurrence of other types of chronic liver diseases following transplantation is controversial. Recent case reports have suggested that both primary biliary cirrhosis¹³ and sclerosing cholangitis¹⁴ recur following transplantation. However, the histological and radiologic findings in these cases were also consistent with chronic rejection and/or ischemic injury to the donor organ.

Immunosuppression. Since 1986, all of our patients have been treated with "low dose" triple immunosuppressive therapy consisting of prednisone, azathioprine and cyclosporine. Cyclosporine has revolutionized liver transplantation and is singularly responsible for the improvement in long-term survival observed during the past decade. This agent, however, is also quite toxic and has a narrow therapeutic window.

The most predictable side effects of cyclosporin are hypertension and nephrotoxicity, although many other side effects ranging from neurotoxicity to hepatotoxicity have been reported.⁶ Renal insufficiency has developed in 55% of our patients and 86% require antihypertensive medication. For treatment we use a variety of agents but usually avoid diuretics, since this may exacerbate renal insufficiency.

Several different assays are available to monitor cyclosporin levels. However, marked intra- and inter-assay variation make it very difficult to interpret cyclosporin levels from different laboratories. Thus

our patients mail blood samples drawn by their home-town physicians to our laboratory for cyclosporine determinations. Doses are then adjusted by phone as needed. Such close monitoring of cyclosporine levels by a single laboratory coupled with the use of two other immunosuppressive medications may explain why we have not seen chronic rejection in any of our patients during the past four years.

Long-term immunosuppression also carries with it a continued risk of serious bacterial, fungal and viral infections. Minor influenza infections appear to be as common as seen in the general population, although resolution of flu-like symptoms seems to take longer. The most common troubling viral infections include cytomegalovirus and herpes viruses. Both can cause elevations in serum AST, ALT and bilirubin, and liver biopsy is frequently required to ensure that rejection is not present. Both respond to appropriate anti-viral therapy, gancyclovir and acyclovir respectively.

The increased incidence of malignancy in chronically immunosuppressed patients is also a major concern and includes carcinoma of the skin and lip, lymphoma and Kaposi's sarcoma. In our program one patient has developed skin cancer two years after transplantation.

Financial Considerations

The cost of liver transplantation is substantial, ranging from \$100,000 to \$250,000 or more. This figure is affected by the patient's overall clinical condition at the time of transplantation, which influences the risk of post-operative complications and the length of hospital stay. These enormous costs can threaten the financial stability of almost any family. Fortunately, most major insurance carriers now recognize the benefits of hepatic transplantation and pay at least a portion of these costs. To assist the patient with these complex financial considerations and to define the remaining financial obligation each patient will incur, a hospital financial administrator negotiates with the insurance carrier on behalf of each patient at the time of the transplant evaluation. With the help of such negotiations approximately 20% of our patients have discovered that their medical insurance fell short of their expectations and have had to raise money before a transplant operation could be performed. This is another reason why early referral is important.

Liver transplantation has recently been recognized by Medicare as an acceptable form of treatment for chronic liver disease and will soon be covered by this program. Thus lack of medical insurance is no longer an excuse for not referring an otherwise excellent candidate for transplant evaluation.

Summary

Over 100 liver transplant procedures have been performed at the Medical College of Virginia since 1984. Overall survival has ranged from 70-80% during

the past three years. Survival declines considerably in persons who have had numerous complications of chronic liver disease, and with significant deterioration in hepatic synthetic function. The single most important factor in determining patient survival is early referral of patients with chronic liver disease for transplant evaluation. Proper timing for transplantation can then be determined by the transplant team working in close association with the patient's referring physician.

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Mapping Out Skin Cancer: Mohs Micrographic Surgery

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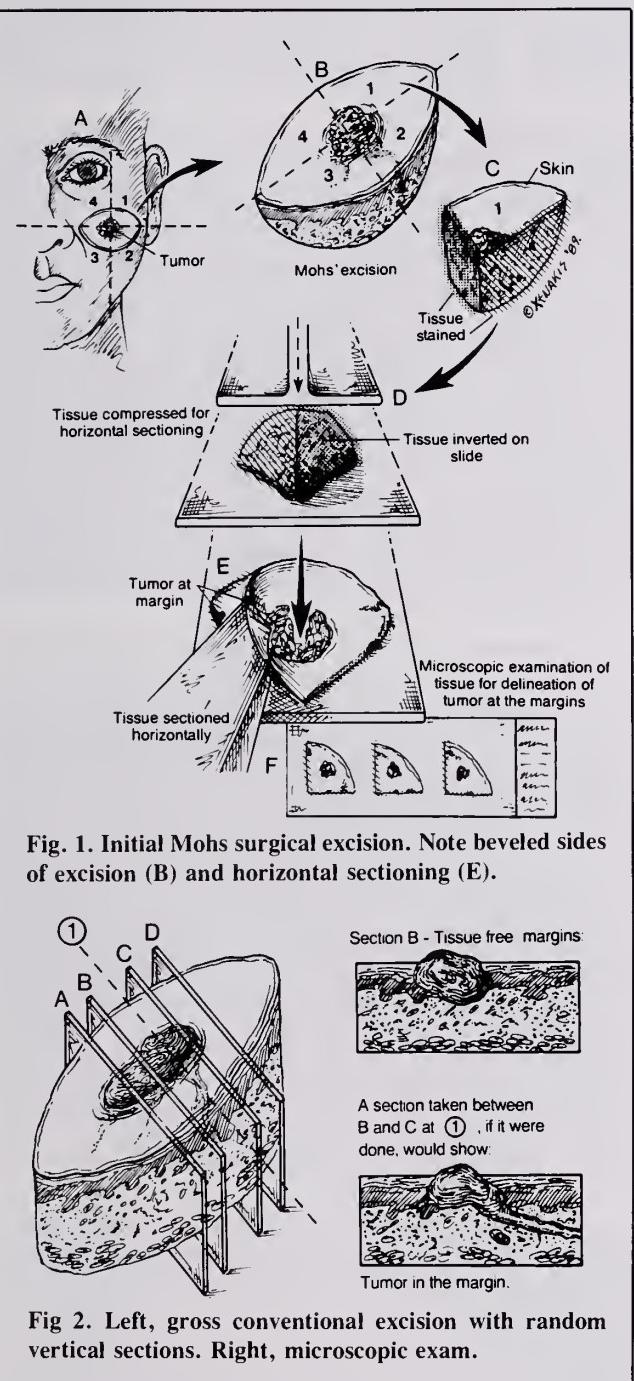


Fig. 1. Initial Mohs surgical excision. Note beveled sides of excision (B) and horizontal sectioning (E).

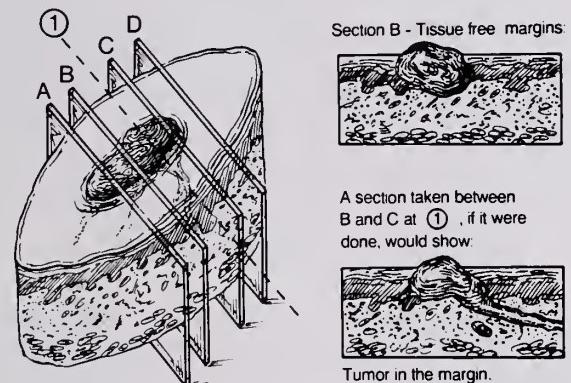


Fig. 2. Left, gross conventional excision with random vertical sections. Right, microscopic exam.

ALTHOUGH it is impossible to know the exact incidence of skin cancer in the United States, it is recognized as being our most common malignancy. The general incidence, probably underestimated, is often quoted as 500,000-600,000 new cases per year.¹ Of these, basal cell carcinoma (BCC) is much more frequently encountered. The ratio of BCC to squamous cell carcinoma (SCC) varies, depending upon the type of practice and geographic latitude of the reporting authors, but usually is thought of as being 6:1 to 10:1. Fortunately, malignant melanoma (MM) is the least common of the three commonly seen skin cancers, and accounts for an estimated 27,000 new cases per year.²

The incidence of non-melanoma skin cancer is felt to be rising, as is the incidence of melanoma. From the 1960s to the 1980s the incidence of SCC has increased almost three times, and that of melanoma four times.³

There are a number of well-recognized and accepted methods used in the treatment of skin cancer. The most commonly employed therapeutic modalities include curettage and electrodesiccation (not for melanomas); excisional surgery; radiation therapy (not as primary therapy for MM); and cryosurgery. Each of these methods requires appropriate patient selection.⁴

Cure rates for primary non-melanoma skin cancer using these modalities varies from 77% to 98%, depending upon the method chosen, tumor type and location, and reporter's special interest and inherent bias.⁵⁻⁷ It is well recognized that the cure rate for recurrent tumors is significantly lower and may only approach 60%.⁸ It is also appreciated by those who treat skin cancer on a regular basis that certain histopathologic subsets of tumor types (such as morphea-like basal cell carcinoma, sclerosing basal cell carcinoma, and basosquamous carcinoma) and those tumors in certain anatomic locations (inner canthus, nasolabial, and periauricular sulcus) predispose to higher recurrence rates. Certain subsets of the patient population (younger patients and in particular, young women) may also be at increased risk for tumor recurrence.^{5,9}

Mohs surgery, also known as Mohs micrographic surgery or microscopically controlled surgery, is a very precise method of excising skin cancer (Fig. 1), affording cure rates of 99% in primary basal cell carcinomas and 95-96% in the more difficult or recurring lesions.¹⁰ This method is not new. It was developed by Dr. Frederic Mohs at the University of Wisconsin about 45 years ago. The technique (Table 1) is time consuming, labor-intensive, and is performed by only a relatively few physicians because of the requirements for special facilities, personnel, and training. These requirements may necessitate an increased cost for tumor removal when compared to simple excision. However, for many tumors, this increased cost is offset by the high cure rate (negating the need for subsequent treatments) and preservation

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Table 1. Steps Involved in Mohs Micrographic Surgery.

1. Locally anesthetized tumorous area thoroughly curetted to debulk and help delineate margins.
2. Reference map drawn corresponding exactly to patient's lesion and showing important adjacent anatomic landmarks.
3. Grid-like identification marks drawn over area to be excised and drawn in on reference map.
4. Saucer-like excision performed.
5. Excision divided into appropriate-sized sections, each assigned a number, and sections replicated on map.
6. Contiguous edges dyed in different colors and indicated on map.
7. Tangential frozen sections performed on each section in Mohs surgery laboratory to encompass undersurface of specimen and entire peripheral (epidermal) margin.
8. Sections stained and examined microscopically by the Mohs surgeon.
9. Location of any residual tumor plotted on map.
10. With map adjacent to patient's surgical wound, exact location of residual tumor can be determined. If tumor is still present, another thin layer, encompassing the "tumor-positive" area, is excised and processed as above. This procedure is repeated as many times as necessary until tumor-free margins are achieved.

of normal tissue and function.^{11,12} Compared to excision in an operating room with "frozen section control," Mohs surgery is extremely cost effective. Mohs micrographic surgery units currently exist at, or in affiliation with, the three Virginia medical schools.

Micrographic vs Excisional Surgery

Conventional excisional surgery is performed with scalpel blade cutting perpendicular to the skin's surface. Regardless of the geometric design of the excision, the removed specimen has vertical sides, or edges. It is impossible to examine the tissue margins microscopically in their entirety unless one cuts vertical sections around each edge and cuts a horizontal section across the entire specimen base. The most common method of "checking margins" is based upon some form of "bread-loafing" (Fig. 2). Using this method, a number of vertical slices (generally 3-5) are taken from the specimen and examined histologically. What about the intervening tissue that is not removed for examination? In a specimen 1.0 centimeters in length, there would be, depending upon the thickness of the slice removed for microscopic examination, 2000 slices (if they were 5 microns in thickness). An analogy to this would be to take a book and stand it on its binding. One can readily appreciate the lack of information that would be obtained by removing only 4 or 5 of the pages for examination. If one were to take several slices, or pages, from the ends and the center, it would enhance our knowledge of the contents but still leave a lot to be desired. What about all the other slices. Do they contain tumor, and does it extend to the margins?

Fortunately, most primary skin neoplasms are solid in nature (like a pea or bean in the skin) and repre-

sentative sections prove to be adequate in a large percentage of cases. However, some types of tumors have tentacle-like strands of tumor cells extending beneath the surface in a subclinical manner. These tumors are often recurrent tumors and morphea-like basal cell carcinoma. This same growth pattern is also frequently seen in scars and in certain anatomic locations, especially in embryonic fusion planes such as the inner canthus, the nasolabial and periauricular sulci, and where the overlying skin is bound down to an underlying structure, such as the cartilage of the ear or the ala nasi. Unfortunately, in these situations conventional "margin checks," whether by frozen section or paraffin-embedded sectioning, frequently prove to be inadequate.

Indications for Mohs Surgery

In this author's experience the major indications for Mohs micrographic surgery are as follows:

1. Recurrent tumors, for reasons already cited in the text. The cure rate by other methods is less than satisfactory.
2. Certain anatomic locations have already been alluded to and include nasolabial sulcus, inner canthus (Fig. 6), periauricular sulcus, ala nasi, concha of ear. These areas have higher than "normal" recurrence rates.
3. Tumors with poorly defined margins (Fig. 3) could best be treated with Mohs surgery. If the margins are not well-defined grossly, it adds to the chance of inadequate treatment.
4. Certain histopathologic types: morphea-like (sclerosing) BCC and basosquamous carcinoma. The sclerosing BCC is notorious for resisting initial treatment attempts as it is poorly defined and beneath the surface has subclinical extension with pseudopodic strands of tumor cells. Basosquamous carcinoma is invariably more invasive than its appearance indicates.
5. Large or aggressive tumors (Fig. 5). These may be primary or recurrent in nature. It should be noted that Mohs surgery results in the smallest adequate excision while simultaneously offering the highest cure rate. This maximal conservation of normal tissue allows it to be well suited for lesions in important anatomic areas where one wishes to minimize the extent of surgery and subsequent scar. This is of particular value in younger persons who may be at increased risk of recurrence (see Cases 1 and 2).

Tissue conservation is also important for cancers on the penis or digits (Fig. 4), where the only alternative may be amputation.

Case Reports

Case 1. A 39-year-old woman was referred for a poorly defined basal cell carcinoma overlying the left lateral eyebrow. She was born and raised on a farm in the South and had had one previous skin cancer.



Fig. 3 (upper left). Poorly defined and recurrent basal cell carcinoma of nasal tip.

Fig. 4 (upper right). Squamous cell carcinoma of right index finger.

Fig. 5 (lower left). Recurring squamous cell carcinoma of postauricular region.

Fig. 6 (lower right). Sclerosing basal cell carcinoma of medial canthus.



Fig. 7, Case 1. Upper left, preoperative view of 39-year-old woman with poorly defined basal cell carcinoma of left suprabrow. Upper right, postop defect after four conservative excisions. Lower left, side-to-side closure. Lower right, followup at 10 weeks.





Fig. 8, Case 2. 25-year-old man with squamous cell carcinoma of lower lip.
Top, preoperative appearance of granulomatous lip lesion.
Middle, postoperative appearance after three surgical excisions using Mohs micrographic surgery.
Bottom, appearance at four months with minimal distortion after healing by second intent.

Clinically, the lesion was poorly defined and measured approximately 1.5×2.0 cm (Fig. 7A). Initial excision, after curettage, revealed tumor extension in medial and inferior margins. The "positive" areas were sequentially excised (total of four excisions) until a tumor-free defect was achieved, measuring 2.0×2.5 cm (Fig. 7B). The defect was closed primarily in a side-to-side manner (Fig. 7C), and early (10-week) postoperative results were acceptable to both the patient and myself (Fig. 7D).

Case 2. A 25-year-old man presented himself with a nonhealing lesion of the lower lip of three months duration. His skin was of a Type I. With the exception of cigarette smoking, no etiologic possibilities were noted. Examination revealed a 1.5×1.0 cm granulomatous, indurated lesion of the left lower lip (Fig. 8A). No regional adenopathy was palpable. A wedge biopsy confirmed this to be a squamous cell carcinoma. The patient underwent Mohs micrographic surgery in three stages (three excisions) before a tumor-free plane was achieved. The subsequent defect measured 3.0×1.5 cm (Fig. 8B) and was allowed to heal by secondary intention, using daily wound care. Healing was uneventful over a four-week period, leaving minimal deformity at four-month followup (Fig. 8C). He has been followed closely for one year with no evidence of local recurrence or regional metastasis. The nature of his disease warrants close followup for the first five years, then periodically (1-2 times per year) indefinitely. He was advised to cease cigarette smoking, which he did.

Conclusion

Mohs micrographic surgery is a highly effective method of surgically removing neoplasms with complete microscopic examination of the removed tissue. The result is maximum preservation of normal tissue and high cure rates even for difficult-to-cure skin cancers. It is generally performed under local anesthesia as an outpatient procedure. Extremely difficult cases because of extent or location may require a team approach.¹³ Rarely, extensive cases may require inpatient management due either to extensive tumor involvement or medical frailty of patient.

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New for Gallstones: Video Laparoscopy for Cholecystectomy

Stephen B. Edge, MD,
B.D. Schirmer, MD, and Janet Dix, PA,
Charlottesville, Virginia

GALLSTONE disease is a serious public health problem, accounting for over 750,000 hospital admissions and 450,000 operations per year in the United States. Until recently, cholecystectomy by laparotomy was the only treatment available for symptomatic cholelithiasis. While fully effective and safe, open cholecystectomy (OC) entails significant pain and discomfort with disability lasting 4-6 weeks. Alternative therapies for cholelithiasis include extracorporeal shock wave lithotripsy (ESWL), dissolution therapy with oral ursodeoxycholic acid or methyl tert-butyl ether gallbladder irrigation.^{1,2} These therapies are markedly less effective in treating gallstones than OC and experience to date suggests they will not have the broad application initially hoped.¹⁻⁴

A new technique, laparoscopic cholecystectomy (LC), allows cholecystectomy without laparotomy. LC was first performed in France in 1987 and in the U.S. in 1988-89.^{5,6} To date, an estimated 2000 patients have had LC in the U.S. Advantages of LC over OC include diminished postoperative pain and disability. Most patients are discharged within 24 hours of LC and return to full activity and employment within one week. Thus LC retains the full efficacy of cholecystectomy without the drawbacks of pain and economic loss to the patient.

This paper reports the initial experience of the LC program at the University of Virginia. We also make suggestions and offer guidelines for other institutions planning LC programs.

Materials and Methods

Training: SBE and BDS received LC training at Mount Sinai Medical Center in Chicago, Illinois. The three-day course, sanctioned by the Society of American Gastrointestinal Endoscopy Surgeons (SAGES), included didactic sessions and extensive animal laboratory experience (porcine model). Further animal experience was obtained at the University of Virginia

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to familiarize OR staff. The first ten procedures were performed with both BDS and SBE in attendance.

Technique: General anesthetic with endotracheal intubation are used with bladder and stomach drainage. With a CO₂ pneumoperitoneum, a 10-mm trochar is placed below the umbilicus into the abdomen. After inspection for other pathology and adverse conditions, a second 10-mm trochar is placed at the xiphoid, and accessory 5-mm trochars placed just above the anterior superior iliac spine and below the costal margin (Figure 1). The gallbladder (GB) fundus is retracted

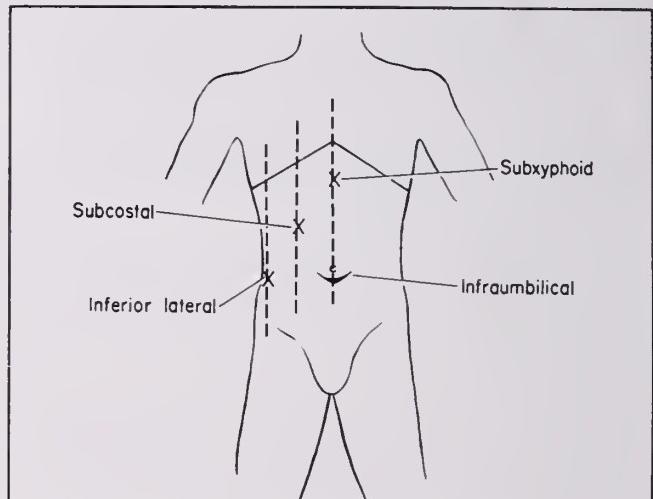


Figure 1. Laparoscopic trochar placement for cholecystectomy. Telescope through infraumbilical trochar, working instruments through other three trochars. Fifth trochar occasionally used to aid duodenal retraction.

superiorly to expose the porta hepatis and fixed in place. The neck of the GB is retracted laterally through the subcostal trochar. Using insulated scissors, hook and spatula LC instruments with cautery through xiphoid trochar, the cystic duct and artery are dissected free. A clip (Endoclip, Autosuture, Inc.) is applied to the cystic duct flush with the GB. Cholangiogram is performed through a 6F whistle tip urethral catheter placed in the cystic duct. The catheter is removed, the proximal cystic duct clipped and divided, and the cystic artery clipped and divided. The GB is dissected off the gallbladder bed with electrocautery. The GB is drawn into the infra-umbilical trochar and brought through the abdominal wall. The skin is closed and the wounds injected with bupivacaine (Marcaine®).

Patients: From February 23 to April 16, 1990, 20 patients underwent laparoscopy for cholecystectomy at the University of Virginia. All had symptoms of gallstone disease and proven cholelithiasis by ultrasound. Consent was obtained under an investigational permit approved by the Human Investigation Committee of the UVa Health Sciences Center.

Results

We attempted LC on 20 patients. Age ranged from

23-84 years with 16 females. Weight ranged from 50-100 kg. Four had a single large stone, Three had two stones and 13 had three or more stones.

LC was successful in 18/20. Cholangiography was attempted in 14 and successful in nine. No common duct stones were found. Two anatomic anomalies identified included a short cystic duct and an accessory right hepatic artery. Two patients (10%) were converted to open cholecystectomy at surgery, one for a retractor tear in the gallbladder which could not be controlled and the other because the cystic duct was too short for clip closure. Both recovered without complication. Nineteen of 20 patients were admitted the day of surgery. Of those who completed LC, 16/18 were discharged the day after surgery eating a regular diet. Nausea was common the day of LC but resolved by morning. Analgesia use was minimal with most only taking oral medication. Five out of 18 required no parenteral narcotics after leaving the recovery room. All LC patients returned to normal activity within one week of the procedure.

Two patients had intraoperative complications. One patient developed extensive subcutaneous emphysema near completion of LC with pCO_2 of 95 and pH 7.06. LC was completed with rapid resolution of blood gas abnormalities and subcutaneous emphysema. The second complication was a gallbladder tear during extraction in our second patient, spilling stones onto the omentum. The debris was removed piecemeal and the patient recovered without incident.

Discussion

Laparoscopic cholecystectomy is a new procedure with marked benefits over other treatments for symptomatic cholelithiasis. While retaining the complete efficacy of OC, LC reduces hospital stay, perioperative pain and disability and overall cost. It is likely to replace 60-80% of open procedures.

LC appears safe, but must measure up to the proven safety of OC.⁷ Bile duct and arterial injuries are grave complications of cholecystectomy which might occur more frequently with LC if anatomic anomalies go unrecognized. Our early experience and that of others indicates that the LC dissection is safe.

The reduction in perioperative discomfort and disability with LC make cholecystectomy a more palatable option. This may further limit the use of ESWL and dissolution therapy. ESWL is most effective for single stones <20 mm. In the Dornier study, 50% were stone-free one year after treatment (combined with ursodeoxycholic acid).³ With two or three stones, success is less likely (22% stone-free at 6 months). Patients with >3 stones have not been treated. Patients treated with ESWL are at risk for stone recurrence. Oral dissolution therapy is effective in about 30% of patients, requires up to two years of treatment, and costs \$4 per day.^{2,4} It is most effective in those with very small single stones. Stones recur in at least

50% of patients within 2-5 years, requiring retreatment or cholecystectomy.

The indication for LC is symptomatic cholelithiasis. The reduced discomfort of LC should not extend the indications for cholecystectomy to include asymptomatic gallstones. There are no absolute contraindications to LC except anesthetic risk and bleeding disorders. Relative contraindications include extensive previous upper abdominal surgery, a scarred, contracted gallbladder obscuring anatomic detail, and acute cholecystitis. However, LC has been performed in all these settings. An initial attempt at LC with conversion to OC if needed is now our standard practice. All patients must be informed with signed consent of the possibility of intraoperative conversion to OC.⁶ In our initial 20 patients, two (10%) were converted to OC.

We use monopolar electrocautery for hemostasis and dissection. Other groups use bipolar cautery or laser (KTP or contact Nd:YAG). There is no advantage of any dissection technique over another. Electrocautery is readily available and easy to use. Laser adds significantly to both the startup and ongoing cost of the procedure without added benefit.

LC is gaining rapid acceptance in the surgical community. At the time of this writing, four centers in Virginia have performed LC. The procedure requires special training and equipment. Training in laparoscopic surgery is now an integral part of our residency training program. For surgeons already in practice, significant time and effort is needed to secure adequate training. Courses offered for practicing surgeons vary in "hands on" experience from a single procedure in the guinea pig to numerous procedures in 30-40 kg pigs. Requirements for course sanctioning by SAGES include participation in six porcine LC procedures (two each as camera operator, 1st assistant and surgeon).⁸ The course offered at the University of Virginia includes participation in nine porcine procedures.

Institutions need to define training and credentialing criteria for practicing surgeons to ensure safe introduction of the procedure. Reasonable credentialing guidelines include 1) full training and privileges in biliary tract surgery; 2) completion of an LC course with sufficient animal experience as outlined above; 3) demonstration of diagnostic laparoscopy skills; 4) preceptorship for 3-5 cases by a surgeon experienced in LC; and 5) LC to be performed by two surgeons with knowledge of the procedure (senior resident or second staff).

The widespread use of laparoscopy in general surgery will surely herald the development of other applications of minimal access surgery as it has in gynecology. Laparoscopic appendectomy is possible in many cases and at least one investigator has performed laparoscopic parietal cell vagotomy.⁹ A clip for internal inguinal ring obliteration is under development for laparoscopic herniorrhaphy.¹⁰

Compulsory Medical Education: The Downside

TO ENTER the profession of medicine is to embark upon a lifetime commitment to education. In that sense, continuing medical education (CME) is simply an expression of that commitment. In 22 states, continuing medical education credits are now required for licensure (Virginia is not among them); many medical societies require CME credits for membership (including the Medical Society of Virginia); and most specialty boards require CME credits for recertification.

We are beginning to see the undesirable side effects of mandatory continuing medical education. By this time we all passively accept the increased record keeping, the outrageously high cost of acquiring the appropriate credits, the hypocrisy involved in taking a cruise or vacationing on a tropical isle while taking a course for CME credit. I personally have lost the embarrassment of asking whether I will be getting CME credit for a course, lecture, or seminar, rather than just wondering whether it will be interesting or beneficial to me. It seems the dim past, although it is only a few years ago, when we physicians could choose on our own what was necessary to further our education.

Compulsory CME is simply not the best way to further a physician's growth in knowledge and ability. It uses only the negative connotation of punishment, rather than any true positive reward. Yet we have seen a whole new industry developed for providing, documenting, and judging of CME, all this at a high cost to be paid for in state and national society dues, in the cost of the meetings, and in the extra work and documentation in our offices. Of course this expense is passed on to our patients. Although there is evidence that knowledge is gained from CME (this of course has always been true), there is no evidence that it significantly alters practice. Without ever more complex and onerous controls, how does one deal with someone who arrives late at a meeting or leaves early, who daydreams through the program, or who takes a brief

catnap, particularly in postprandial or evening meetings? How do we prove that what was presented was learned? How do we prove that what was learned was applied? How do we prove that what was applied alters outcome? I am not arguing against altered (improved) outcomes, nor the fact that increased knowledge is a factor (but not the only factor) in improved outcomes—only the means of proving it via forced education.

There is an implied assumption, based on the methods of our schooldays, that most learning comes from a formal setting wherein a teacher passes on knowledge to a student who supplements the classroom knowledge with "homework." Although I am sure that this continues to hold true to some extent throughout the clinical years, I doubt that this makes up the bulk of applied knowledge. Methods of learning are highly individualized. Some learn best in solitude, others in a crowd. Some need background noise (music or voices), some need complete quiet. Some learn best by visual means (watching a tape, reading a book, or direct observation), some by oral means (discussions, seminars), some by aural means (lectures, cassette tapes), and most by a combination of these. It is my strong suspicion that far more applied clinical knowledge is learned in hospital corridors and nursing stations, doctors' lounges, and hospital cafeterias and in telephone discussions of patient problems than by any formal CME program. (This is probably not true of basic knowledge, which must be transmitted in a more formal setting.)

ONE VIEW expressed early on was that if we did not do something about policing our profession, the government would. Well, we have done something, and Congress, in the person of Rep. Fortney "Pete" Stark, has responded in a predictable fashion. He has introduced HR 4464, which, if passed (not likely for now but a portent for the future), would mandate that physicians would have to take a profi-

Summary

Laparoscopic cholecystectomy is a new technique for treatment of symptomatic cholelithiasis. Advantages of efficacy, reduced pain and shortened disability make this the preferred treatment. The procedure can be learned by surgeons versed in biliary surgery. Physicians not fully trained in biliary tract surgery should not perform LC because of the dangers of bile duct and arterial injuries, and because of the need for immediate open surgery in 5-10% of patients.

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ciency examination if they are to be permitted to treat Medicare patients (*Am Med News*, 4 May 1990). So much for the theory that lawmakers could be held off if we embarked on self-policing.

Our professional organizations ought to insist that if repeated testing is the measure of proficiency, then Congress ought to lead the way. Not only members of Congress but their legislative assistants ought to be required to be examined periodically on U. S. history, the U. S. Constitution, and constitutional law, political science, economics, and, most importantly, ethics. Those who fail the testing should be forced to give up their positions and leave Washington so they cannot end up with lucrative "consulting" positions, all too often a euphemism for influence peddling.

There is another proposal I would make. On the 10th anniversary of graduation from medical school and every ten years thereafter, I propose a compulsory return to school of approximately three weeks. Although beneficial from an educational standpoint (for example, the first week could be devoted to basic science, the second to general clinical medical advances, and the third to advances in one's own and related specialties), the basic purpose would be renewal for the physician. This would be both physical and mental renewal. Activities outside the formal classroom environment would include basic health parameters performed on each physician, with followups to see that corrections are taken; organized discussions of subjects ranging from medical ethics to impaired physicians to practice problems to health policy matters; and of course renewing old acquaintances in a relaxed and informal environment. This could give each physician the psychic energy to face the coming decade.

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who have accomplished the course prescribed for the degree
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the President of the University and the Dean of the
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have conferred the degree of

Doctor of Medicine

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Robert Lawrence White

who have accomplished the course prescribed for the degree
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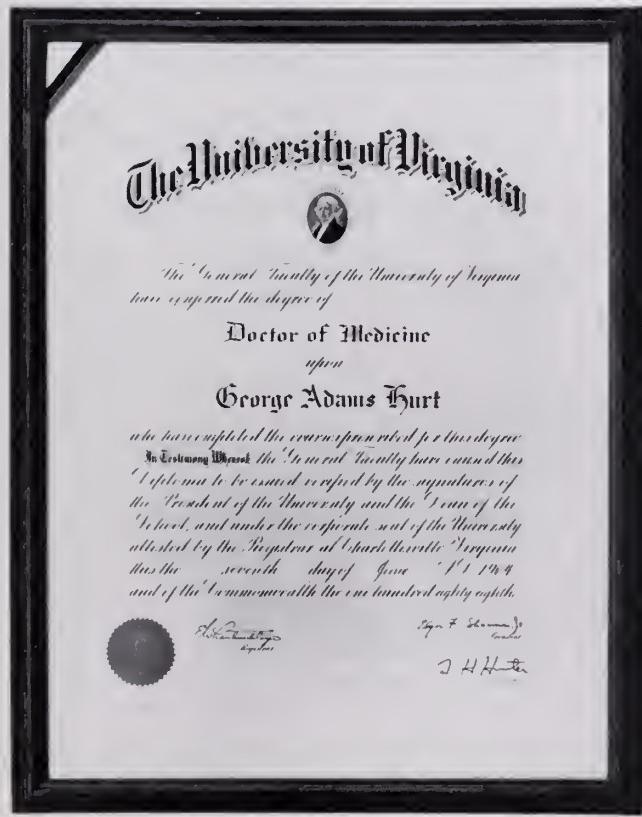
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Avoidable Claims in the Liability Experience of Virginia FPs Insured By the Reciprocal

Jen Lee Nelson, MHA, and
Barbara S. Brown, PhD,
Richmond, Virginia

A STUDY of the Virginia Insurance Reciprocal (TVIR) liability claims experience for family physicians was conducted to summarize the frequency and severity of claims. Claims adjudicated between 1981 and 1989 were reviewed to provide an understanding of physician and claimant characteristics, areas of greatest risk and loss, and allegations made. Another goal was to identify factors that could prevent malpractice claims against family physicians. The analysis illustrates the impact risk management strategies can have in reducing malpractice losses.

MATERIALS AND METHODS

Claims filed from January 1, 1981, through December 31, 1989, were reviewed. During the nine years, 120 claims were filed against family physicians insured by the Reciprocal. Of these 120 claims, 79 (66%) were closed as of December 31, 1989, and 41 (34%) were still open on that date. The files for all identified closed claims were analyzed.

All claims were made against Virginia physicians. The Reciprocal insures an estimated 37% of the family physicians in Virginia, of which approximately 15% were involved in a claim during the study period.

The files of 73 of the 79 closed claims were available and were abstracted to identify information on the frequency and cost of claims. Claims with and without payments were included. Each claim represented one occurrence. For each occurrence, more than one physician could have been sued; however, the claim was treated as one case. In addition a physician may have been sued for more than one incident within the study period.

Information collected included: claimant's age, sex, occupational status at time of alleged injury; physician's type of practice (solo or group), board certifi-

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cation, number of years in practice at time of alleged injury; location where alleged negligence occurred.

The allegations of negligence made in each claim were grouped into seven categories. Risk management issues were identified from information recorded in the claims reports and other summary information in the files. A cost analysis was done for all closed claims. Claims payments and litigation costs associated with each claim were noted. Dollar figures were adjusted using the U. S. City Average Medical Care Price Index as the conversion factor.

A summary analysis was done to determine which claims might have been avoided if certain risk management strategies had been implemented.

RESULTS

The Claims

The 73 claims involved 66 family physicians, which is approximately 15% of the family physicians insured by the Reciprocal and an estimated 5% of the total family physicians practicing in Virginia in 1989. The analysis showed that 41% of the closed claims involved an insured physician and at least one other health care provider, and 44% of the claims were against individual insured physicians only. The remaining 15% represented the closed claims involving a family physician insured by TVIR and a provider insured by another liability carrier.

As of December 31, 1989, 36 claims (49.3%) had been closed with indemnity payments made on behalf of the physician and 37 claims (50.7%) were closed with no payment. This is consistent with national trends.¹ Table I shows the number of paid claims and total indemnity payments by payment ranges.

Table I. Payment Ranges of 36 Paid Claims Against Family Physicians Insured by the Virginia Insurance Reciprocal 1981-89.

Payment Ranges	Number of Paid Claims	Total Actual Indemnity Payment
\$1 to \$999	1	\$ 151
\$1,000 to \$4,999	5	11,935
\$5,000 to \$9,999	6	46,500
\$10,000 to \$24,999	8	120,699
\$25,000 to \$49,999	2	57,500
\$50,000 to \$99,999	4	272,500
\$100,000 to \$249,999	7	1,000,000
\$250,000 to \$499,999	0	0
\$500,000 and Greater	3	2,087,060
Total	36	\$3,596,345

Year of payment, frequency and total adjusted payment (inflation factor applied) for all closed claims are shown in Table 2.

The length of time elapsing between the alleged injury to the filing of a claim ranged from less than one month to 48 months (four years). The time period most frequently noted was 19-24 months. Five percent of the claims were filed within the same month the alleged injury occurred, and about 8% of the claims took two years or longer to be filed.

The length of time from filing of the claim to

complete disposition ranged from greater than one month to 76 months (6.3 years). The majority (79%) of the claims made against family physicians were resolved within two years of filing.

The principal allegations of negligence were identified and are shown by category in Figure 1. As Table 3 shows, medication- and treatment-related allegations had the highest total payout of all allegation categories. The single obstetric-related claim accounted for \$827,108, or 18% of the total payout for family physicians during the study period.

The Physicians

Of the 66 family physicians involved in claims during the study period, 60 had a single claim made against them, five had two claims, and one physician had three claims within the 9-year period. The fact that the majority (91%) of the physicians had only one claim against them suggests that claims activity does not identify, or is not necessarily linked to, poor medical practice.²⁻⁴

Of the 73 closed claims studied, 64% involved physicians who were board certified in family practice. This same board certified group accounted for 63.7% of the total payments for these claims.

About 47% of the family physicians were in individual (solo) practice, and the remaining 53% were distributed over groups of varying sizes. The majority of the claims (81%) were concentrated in solo practices and groups of two and three physicians.

Analysis of the number of years that physicians involved in these claims had been in practice at the time of the alleged injury revealed that the largest percentage of claims (60%) represented physicians in the first 15 years of practice. The other 40% of the claims were spread over longer practice ranges.

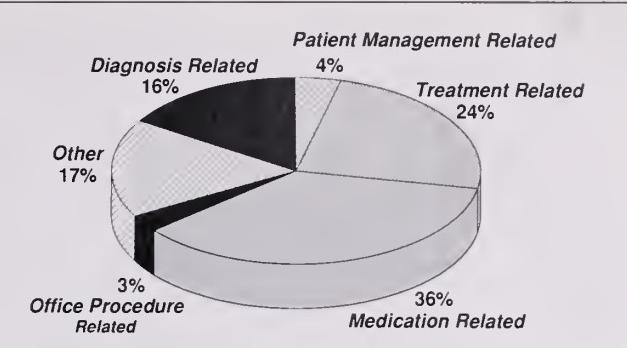


Fig. 1. Percent of Total Payment by Allegation Category of Closed Claims Against VIR-Insured Family Physicians in 1989.

The family physician's office was the setting for 49% of the alleged injuries, and these claims accounted for \$3,402,867 (73%) of the total payout for this specialty. The remaining 51% of the alleged injuries occurred in the hospitals, nursing homes, health departments or prison clinics.

The Claimants

When analyzed for claimant's age, the 73 closed claims appeared to be evenly distributed, with a slight increase in claims in the 40 years and older range. The age range most often represented was 65 years and older (14 claims made). Only 5% of the claims were on behalf of patients injured at birth to 1 year old.

About 53% of the claims were made by females, and 47% by males. Female claimants accounted for \$2,428,781 (52%) of total payments made, males for \$2,205,595 (48%).

Over one-third (37%) of the claimants were employed (self-employed included) at the time of alleged injury, and 27% were either homemakers, dependents, or unemployed. Incarcerated individuals in the state

Table 2. Year, Frequency and Adjustment Payments in 36 Claims Against Family Physicians Insured by the Virginia Insurance Reciprocal 1981-1989.

Payment Year	No. Claims Closed	Actual Indemnity Payment (\$) ¹	Actual Expenses Payment (\$) ¹	Total (\$) ¹	Avg. Payment per Claim (\$) ¹
1984	4	18,931	12,541	31,472	7,868
1985	7	98,764	62,299	161,063	23,009
1986	13	1,446,533	118,122	1,564,655	120,358
1987	12	1,750,904	177,084	1,927,988	160,666
1988	18	563,523	55,918	619,441	34,413
1989	19	275,000	54,757	329,757	17,356
	73	4,153,655	480,721	4,634,376	491,973

Table 3. Payment by Allegation Category in 36 Claims Against Family Physicians 1981-89.

Allegation Category	Number Claims	Percent Of Total Claims	Total Adjusted Payment	Percent of Total Adjusted Payment
Diagnosis-Related	23	32	738,171	15.9
Treatment-Related	20	28	1,101,487	23.9
Office Procedures-Related	12	16	127,444	2.7
Medication-Related	11	15	1,669,098	36.0
Patient Management-Related	5	7	171,068	3.7
Biomedical Equipment-Related	1	1	0	0
Obstetrics-Related	1	1	827,108	17.8
Total	73	100	4,634,376	

¹Dollar amounts adjusted to reflect 1989 dollars

correctional system accounted for 7% of the claims.

Table 4 shows the number of avoidable claims and the total dollar amounts represented by each in five risk management categories.

DISCUSSION

Over the past nine years, the claims experience of family physicians insured by the Virginia Insurance Reciprocal indicates that about half of all claims made were closed with no payment. Loss payments showed high variability, with an average payment of about \$8,000 per insured family physician. It took an average of 13.5 months from the time of alleged injury to file a claim, and an average of 16.3 months from the date of filing to fully resolve a claim. Allegations of diagnosis- and treatment-related errors were made most frequently, and those related to medications were the most costly in terms of paid losses.

The data suggest that board certification is not linked to the incidence of malpractice claims. This is likely due to the fact that most of these insured physicians were board certified, making it a weak indicator of medical expertise.

Physicians practicing in groups of four or more had a lower incidence of claims than those in solo practice. Larger groups are more likely to implement systems to monitor, flag, and followup patient care. In addition, the peer review available in group practices may influence the quality of diagnosis, treatment, and medication regimens.^{5,6}

Physicians in their first 15 years of practice were named more often in the studied claims than those who had practiced longer. Although the data cannot provide conclusive evidence as to why this trend occurred, it can be proposed that the stability of the practice and established relationships with patients influenced this finding. The most common reason for initiating a suit is patient dissatisfaction. The dissatisfaction can stem from poor bedside manner or the patient's perception that the physician was not thorough or did not render adequate care.^{2,3} The cure for dissatisfaction is communication with the patient and thorough and legible documentation of the patient visit.^{2,4,7}

Approximately one-half of the closed claims in this sample occurred in the family physician's office. Such losses should be easier to avert than those originating in a hospital or other health care setting in which the physician has less influence on procedures.

Thirty-six (49.3%) of the 73 claims could have been prevented by implementing effective risk management practices. These claims resulted in a total loss of \$2,878,678 (62.1%) of the total cost of all the claims.

SUMMARY

In response to the threat of litigation, physicians practice defensive medicine. A more effective means of reducing malpractice liability and improving the

Table 4. Avoidable Claims in 36 Paid Claims Against Family Physicians 1981-89

Risk Management	Number of Claims	Total Dollar Amount ¹
Scope of practice and training of office personnel	10	\$ 429,163
Documentation practices and patient communication	6	258,766
Systems to monitor, flag and followup patient care	8	1,343,896
Coordination of care among health care team	7	112,139
Office procedures	5	734,714
	36	2,878,678

¹Dollar amounts adjusted to reflect 1989 dollars

quality of patient care is to practice defensible medicine, i.e., those practice patterns that reduce the potential for claims and increase the likelihood that when claims do arise, they can be successfully defended.³

Unlike hospital-based specialists, family physicians can directly influence a significant portion of their risk of suit. By focusing management time and attention on the factors in their office practices that are linked to malpractice claims activity, family physicians can reduce the likelihood of being involved in claims.

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SAFEGUARDS AGAINST SUIT

Physicians are responsible for ensuring that all employees and allied health personnel are properly trained and credentialed. Problems that lead to litigation occur when the office receptionist or medical assistant gives medical advice over the phone without the knowledge of the physician. Other suits have arisen from injections being given by untrained and unsupervised assistants. Periodic retraining of employees is as important as initial training. Office personnel need continuing education as much as the physician to keep abreast of new issues and maintain and hone skills.

Documentation practices influence the ability to defend a claim. Charts with pencilled notes, correction fluid alterations, no dates or initials on changes, or lost or missing record components give the impression of poor quality care or altered evidence. The medical evidence is put on the stand as much as the physician. From the point of view of the plaintiff's attorney, if it isn't recorded it didn't happen. A system of physician review prior to filing the patient's record is essential to insure completeness and to document patient compliance with recommendations.

Patient communication is the root of most medical malpractice claims. Patients' perceptions are important. The physician and staff have ample opportunity to shape these perceptions. The time spent with the patient, the rapport built during that time, and the feeling of physician accessibility are perhaps the greatest insurance against a liability claim.

The physician has several options for establishing patient rapport. 1) Orient the front office staff to patient service, activities, and concepts, and teach them how to manage disgruntled patients gracefully. 2) Have information readily available for the patient on the practice, what to do when the office is closed, whom to call and the number. Patients need to understand the rotation of different physicians within a practice and who is available to treat them at different times. To avoid a claim of abandonment, provide patients with written information on partners and cover-

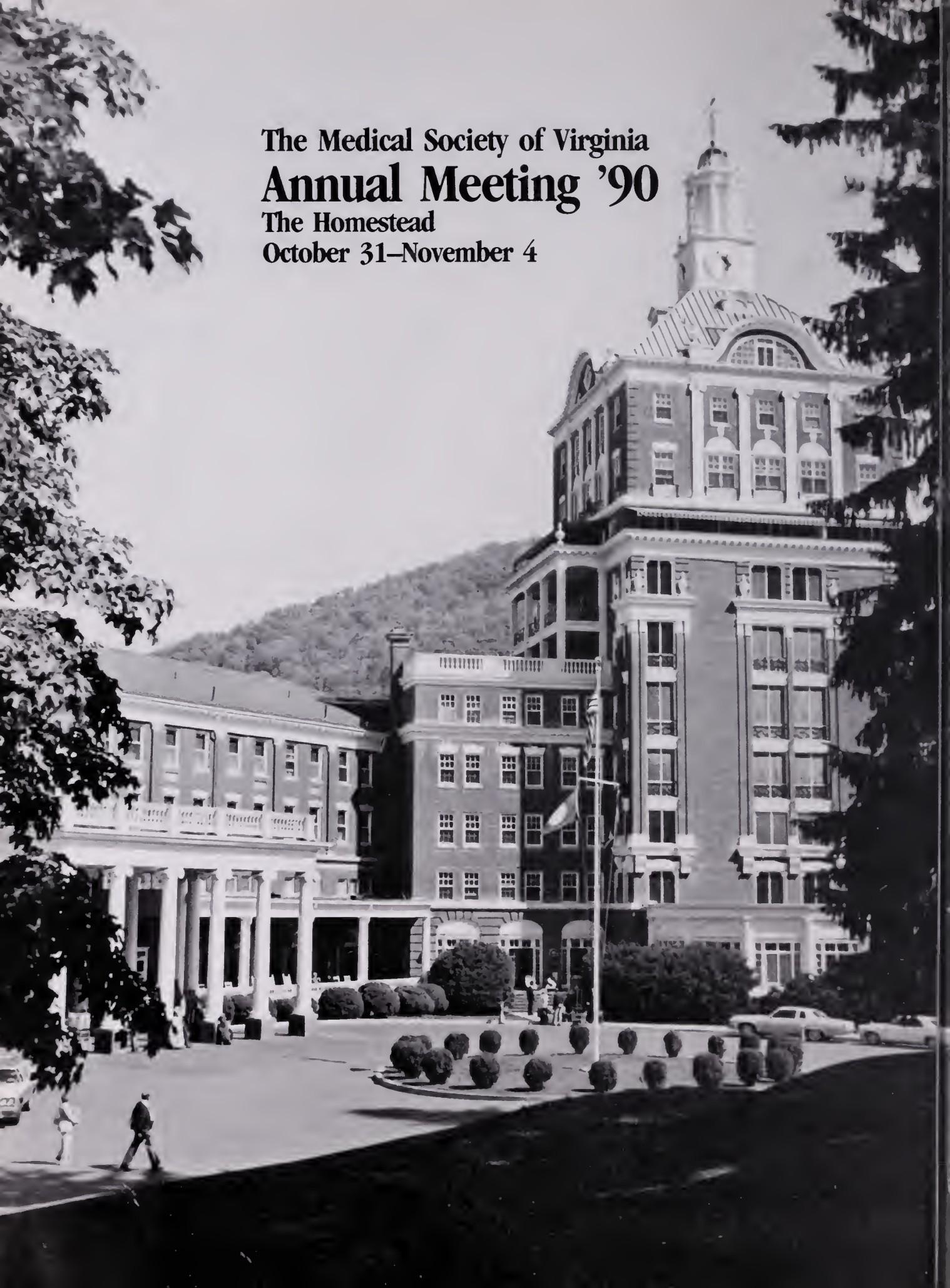
age arrangements and date when the information was received. 3) Billing practices should be clearly explained on the first visit. Much of this information can be covered by implementing a new-patient orientation procedure.

Systems to monitor, flag and followup patient care focus on tracking the receipt of laboratory and other tests, appointments, complaints, prescription orders and refills, phone messages, and noncompliance with medical regimens. It is equally important to get the information or reports on consultations requested from other physicians. Two wrong diagnoses are easier to defend than one! Use of a matrix record for comparing test results and medication regimens over time is recommended for patients with chronic conditions who are likely to have concomitant medical complications of their primary disease and/or to need consulting physicians.

Coordination and continuity of patient care are facilitated by systems or procedures to assure that the office is adequately covered, that patient problems are discussed and documented, and that patient complaints are resolved satisfactorily. If the commitment is made to cover another physician's practice, then the covering physician is responsible for transferring the information necessary for continuity of care. The primary physician is responsible for establishing and maintaining a mechanism for receiving information and documentation from consulting physicians on hospitalized patients.

Office procedures are a key strategy in preventing claims. Protocols outlining procedures for telephone triage, medication refills, scheduling appointments, performance and receipt of test results, billing and problem accounts, termination of the physician-patient relationship, documentation practices, and patient followup are important components of basic office risk management. Information on how to develop these documents is available from medical societies and some liability carriers.

J.L.N. and B.S.B.



The Medical Society of Virginia
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VIRGINIA MEDICAL

Injured Infant Act

THE Injured Infant Act legislated into law by the 1987 General Assembly of Virginia has been the object of considerable criticism by Virginia physicians. Objections include: the narrow definition of the act; the \$5,000 annual fee for participating obstetricians; and the \$250 annual fee for all physicians in Virginia.

A portion of the problem has been resolved: The law has been amended to improve the definition of neurologic impairment. However, the other objectionable features persist.

Indeed, the State Corporation Commission audited

the present birth injury fund and found it inadequate. An assessment order was issued requiring that the insurance industry be assessed one-tenth of one percent of premiums written in Virginia. Expected revenue for this action is \$2.8 million. Currently, the \$250 fee paid by physicians contributes \$1.8 million annually to the birth-injury fund. And all this is in addition to the assessment levied on participating hospitals: \$50 for each delivery, with a maximum of \$150,000.

Isn't it about time for participation by the general public?

E.L.K., JR.

Explain

WE PHYSICIANS are constantly told we must take the time and make the effort to explain the medical problem in detail, yet understandably, to the patient. We must educate the patient so that the patient can then make a rational decision about his or her own life. We are constantly reminded that too many physicians rush their explanations, speak in medical jargon, and befuddle rather than inform the patient. We must explain the concept of wellness to our patients.

A properly given explanation will almost always lead a patient to make a proper decision. Defective decision-making by the patient is a reflection upon the physician. You have to tailor the explanation to the patient and the situation.

My aunt was a heavy smoker for 40 years, between one and two packs a day. Her high blood pressure, her

family history of cardiovascular disease, the advice of several physicians, and the pleadings of her husband and children had no effect upon her cigarette consumption.

About three years ago she had an episode of bloody urine which led to finding a cancerous bladder tumor. Petrified with fear, she prepared herself for the worst. Fortunately, the tumor was removable by transurethral resection.

As she tells the story, she was just barely awake when her urologist came into the recovery room and told her that she had an excellent prognosis. Furthermore, he strongly advised that she stop smoking to decrease the chances of recurrence of her cancer.

And she has not had a single cigarette, nor the desire for one, since that moment. When asked why she had waited so long to stop smoking when she had been

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cajoled and threatened, begged and urged for years, her response was instructive: "No one ever explained it to me that way before."

H. S. CAMPBELL, MD

Lockhart McGuire

ELSEWHERE in this issue is the memoir of Dr. Lockhart Bemiss McGuire. A professor in the University of Virginia, Department of Medicine, Dr. McGuire was beloved by his patients and admired and respected by his students and coworkers alike. And for good reason: Dr. McGuire was the ideal physician, educator, and citizen. His life was modeled after the words of that hymn: "God be in my head, and in my understanding; God be in my mouth, and in my speaking; God be in my heart, and in my thinking; God be at mine end, and at my departing."

E. L. K., JR.

1. Hymn 694, Episcopal Hymnbook 1982. Words by Sarum Primer, 1514

Hard Workers

A RECENT press release from the Virginia Department of Health Professions lists 1,229 complaints recorded by the State's 12 health regulatory boards during the 1989-90 fiscal year. The figure is accurate, but may be misinterpreted. All such complaints are referred to the investigation division, where decision is made as to the validity of the complaint. If the complaint is deemed valid, it is referred for action by the proper board. The actual number of cases received in 1989-90 by the State Board of Medicine totaled 319. Although this is an increase over the 258 complaints in 1985, there does appear to have been a levelling-off of the curve.

During the 1989-90 fiscal year, 340 cases referred to the Board of Medicine were closed. Sixty-four of these were judged to be violations and resulted in sanctions. Eight licenses were revoked, four were suspended, 28 cases resulted in probation and 23 in reprimand. Heading the list were 123 cases involving standards of care. While there were 38 instances of excessive prescribing, it should be noted that in recent years there has been a change in regard to the accepted prescribing of certain drugs; an occasional one of these previously might not have been in violation.

The State Board of Medicine is a hard-working, effective body and one of which the people of Virginia can be justly proud. The Board had functioned well in spite of one major handicap: the present legal system. This is a problem for which there appears to be no solution.

E. L. K., JR.

GUEST SPEAKER

“Holding to Life Lightly But Not Carelessly”

PEOPLE sometimes talk as if God is capricious about who escapes sudden and untimely death. People do not always question the capriciousness of technology and of our economy in all the business of keeping older people alive or in the use of the resources which might have been used with greater benefits in other ways.

One of my more remarkable experiences was joining, in another city, in a family discussion with doctors regarding the use of exciting and miraculous machinery to add an uncertain number of hours, with even more uncertain meaning, to the life of a brother who had already had a long life of love and service plus a year of courageous suffering. He had spent next to nothing on himself, had a wardrobe chiefly consisting of clothing left by deceased parishioners, and frequently refused salary increases. Here the doctors were, proposing measures without mentioning that they would be costing Medicare and other insurance carriers more per day than my brother would have accepted for a long period of hard work.

It would have been a violation of all he stood for.

No matter how little we like to put a price on the care of the sick, the problem is there and becoming more difficult every day. The longer we refuse to face it the harder it becomes.

All of us resist putting a monetary value on a month's or a year's life, without even considering the capacity of that life for continued meaning in this world. Yet the simplest mathematics make it clear that we cannot go on refusing to face the issues involved. We escape by taking refuge in “the sanctity of life” or by saying it is unworthy to talk about cost in such a situation, yet money is both limited and representative of the love, labor and life of others.

This is a most painful tension—the mysterious value of the spark of life against the mysteries of the world's physical suffering in the midst of plenty. Indeed, there is a certain horror about even raising the questions.

How do we decide when we are respecting the sanctity of life and when we have made an idol of not even a whole life but merely some of the “vital signs?”

Most of us are familiar with the quandary over

whether some actions are interventions with natural living or interventions with natural dying. The Richmond Academy of Medicine and the Medical Society of Virginia have promoted a “living will” implementing Virginia's Natural Death Act. I hope each of you has signed one, but there are still many questions about whether the usual form covers enough possibilities. There are also those who point out that a written living will can be limiting by comparison with an understanding developed among members of a family and their doctors. Even then, the trouble is that none of us can decide the way we are going to die, and that brings in the different ways each of us may deal with his own approaching death.

I have always admired the people who have been able to face the approach of death, talk freely with their families about their state of mind and future arrangements (always allowing for the possibility of a reprieve), and yet continue to greet each new day with as much interest and joy as pain will allow.

I suppose I was influenced in these matters by my mother. She paid many visits to hospitals during her last 20 years but always left her bureau in perfect order, with directions for disposal of various items; yet at the same time she lived every day fully. Before her final operation she urged the surgeon to take any chance he wanted to.

I think this is what is meant by holding to life lightly but not carelessly. Yet in this field there remain—and may always remain—terrible questions for which each answer seems to lead to more questions.

We are fortunate that in our day the medical profession is doing its part in *asking* the questions.

JOHN PAGE WILLIAMS, DD

Excerpted from a talk delivered before the Adult Forum at St. Mary's Episcopal Church, Richmond, on March 20, 1988. Dr. Williams was headmaster of St. Christopher's School, Richmond (1940-1951), and then served as Dean of Church Schools in the Diocese of Virginia until his retirement in 1975. Address correspondence to him at 703-G North Hamilton Street, Richmond VA 23221.

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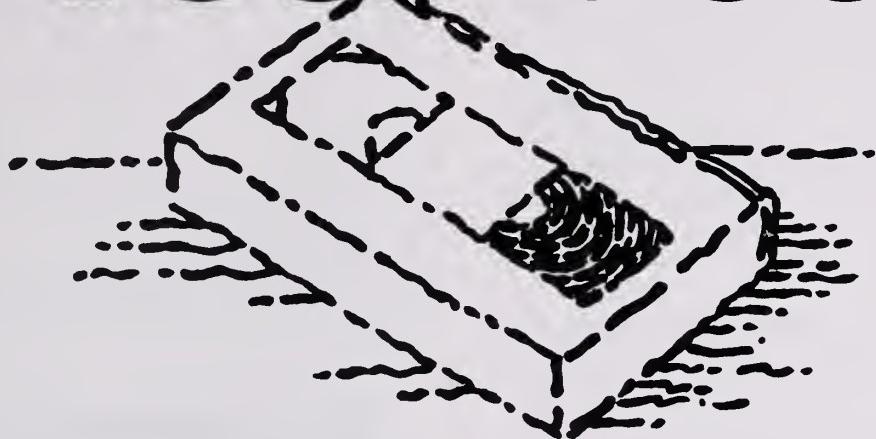
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VIRGINIA MEDICAL OBITUARY

• **Albert Facundo Borges, MD**, Falls Church; University of Havana, Cuba, Medical School, 1943; age 70; died May 17, 1990. He had been a plastic/reconstructive surgeon in Falls Church since 1960.

• **Paul Kiser Candler, MD**, retired general practitioner; Medical College of Virginia, 1936; age 83; died June 23 in Virginia Beach. In private practice in Warrenton from 1946, he joined the Salem Veterans Administration Hospital staff in 1964, serving there for ten years.

• **Thomas Frey, MD**, pediatric ophthalmologist in Northern Virginia and a past president of the Virginia Society of Ophthalmology; Northwestern University School of Medicine, 1958; age 56; died July 10, 1990. He conducted practices in Falls Church and Fairfax and lived in Annandale.

• **Conrad L. Gossels, MD**, internist in Washington and Arlington; Friedrich Wilhelm University, Berlin, 1932; age 82; died August 4, 1990. He retired nine years ago.

• **Tibor Ham, Sr., MD**, pulmonary specialist in Vienna, Virginia; University of Tudomanyegyetem, Budapest, Hungary, 1938; age 76; died July 7, 1990. Dr. Ham left Hungary for the United States in 1951 following the Communist takeover.

• **James Arlington Kirkland, Jr., MD**, general practitioner in Emporia; University of Virginia School of Medicine, 1960; age 55; died July 12, 1990. He was mayor of Emporia from 1972 to 1976.

• **Wilmer Howard Paine, Jr., MD**, Charlottesville; University of Virginia School of Medicine, 1930; age 86; died July 20, 1990. He had practiced general medicine in Charlottesville since 1932.

• **Adney K. Sutphin, MD**, retired Richmond internist; Medical College of Virginia, 1941; age 73; died June 4, 1990.

• **Heda Tibor, MD**, Petersburg; Faculté de Médecine de la Université de Lausanne, Switzerland, 1949; age 76; died February 22, 1990.

Memoir of William R. Hill 1908-1990

By James B. Black, MD, Percy Wootton, MD,
and Wm. Russell Jones, Jr., MD

Dr. Bill Hill, as he was known to many, was a highly respected general surgeon who practiced in Richmond for 49 years, retiring scarcely two months before he died on 10 April 1990.

Dr. Hill was born 4 October 1908 in Statesville, North Carolina, where he was educated in the public



WILLIAM ROWLAND HILL, MD
1908-1990

schools. He received his premedical education at Davidson College and his medical degree in 1934 from the University of Virginia School of Medicine, where he also served as chief resident in general surgery.

Prior to World War II he became associated with Dr. A. L. Herring, Sr., at Grace Hospital in Richmond. He entered the service as a fully trained surgeon and rendered invaluable help to the wounded, serving as major in the Medical Corps with the 8th Evacuation Hospital from the University of Virginia. This hospital served in the North African and Medi-

terranean theaters of operation. After the war he continued his association with Drs. R. C. Siersema and Herring. At the time of his retirement, Dr. Hill was practicing in association with Drs. W. Bruce Stewart, James S. Gregory, Crawford C. Smith, and Jon Palmintier.

Many organizations benefitted at his hand. He was a clinical professor of surgery at the Medical College of Virginia, consultant at the McGuire Veterans Administration Medical Center, and served as president of both the Richmond Academy of Medicine and The Medical Society of Virginia. He was for eight years a member of the State Board of Health and its president in 1979-80.

It would not be possible for me to recall the number of times I have been told of his diligence in visiting patients whom he had once treated but were then situated in another hospital or at home. Such acts of kindness were much appreciated and were performed modestly.

Two days before he died Bill phoned to remind me that a faithful orderly at Grace Hospital had expired and that I should "know about it." His thoughts concerned the welfare of others.

Through the years we often operated together when a patient needed the services of both a general surgeon and a urologist. These occasions were always a pleasant experience, filled with expeditious deliberation and professional good humor. He will be greatly missed from medical practice.

Dr. Hill is survived by his wife, Elizabeth Broaddus Hill of Richmond; two daughters, Sherrard Steele Howen and Beverly Byrd Almond, and one son, John Parrish Hill, all of Charlottesville.

the US Naval Hospital, Chelsea, Massachusetts, he returned to the University of Virginia as chief resident in medicine 1963-1964.

Following his appointment to the faculty in cardiology, Dr. McGuire rose rapidly through the ranks to that of professor in 1974. In 1983 he was named the first Julian R. Beckwith Professor of Medicine. He introduced the concept of the Code 12 (resuscitation) team, the Coronary Care Unit, the Pacemaker Clinic, and the use of the Holter monitor to the University of Virginia Hospital. He was active in the American Heart Association and the American College of Cardiology (governor for Virginia 1973-1976). He devoted a great deal of his time to the American College of Physicians, as member of Council for Virginia, as head of the Clinical Efficacy Assessment Program, and as the recipient of the Laureate Award from the Virginia chapter shortly before his death.

He was elected a member of the Raven Society and of Alpha Omega Alpha in medical school, and of the American Clinical and Climatological Society as a faculty member. He won a citation from the dean for excellence in teaching, and was president of the Society of Fellows of the University.

Dr. McGuire was an active member of the Albemarle County Medical Society (president, 1973); of The Medical Society of Virginia, representing the American College of Cardiology at the House of Delegates; and of the Editorial Board of *VIRGINIA MEDICAL* and the *Virginia Quarterly Review*.

Dr. McGuire cheerfully participated in the Naval



LOCKHART B. MCGUIRE, MD
1934-1990

Memoir of Lockhart McGuire 1934-1990

By George B. Craddock, Jr., MD, and
John A. Owen, Jr., MD

Sir William Osler paid tribute to those physicians who, he said, "have influenced the profession less by their special work than by exemplifying those graces of life and refinements of heart which make character." Such a man was Lockhart Bemiss McGuire, who died April 29, 1990, at his home, of cancer of the colon.

Born in Richmond on May 8, 1934, he was the son of Dr. Hunter Holmes McGuire and Catharine Bemiss McGuire, and brother to Dr. H. H. McGuire, Jr., of that city. He was educated at Episcopal High School and the University of Virginia, where he received the MD degree in 1957. He was trained in internal medicine and cardiology at the Peter Bent Brigham Hospital and Harvard Medical School. After a tour of duty at

Reserve and retired with the rank of Captain, Medical Corps, USNR. He was a former vestry member of St. Paul's Memorial Church in Charlottesville and was a member of the University of Virginia's Center for Clinical Ethics. He is survived by his wife, Anne Kane McGuire of Charlottesville, three children and one grandchild.

He was a loyal friend to colleagues, students, and patients alike. As the ideal physician, educator and citizen, he was a model without equal. To his patients, most of all, he demonstrated skill, devotion, and kindness. He laid less emphasis on the technological advances of cardiology and more on the clinical skills of the meticulous history, the precise comprehensive physical examination and a deep and broad understanding of his patients as individuals grappling with serious problems and needing his kind and patient assistance in decision-making. As he gave of himself to others, he was repaid a thousand-fold by their gratitude, trust, and love.

Shortly before his death, he tried to sum up his life in a typically modest fashion: "Dr. McGuire was the recipient, through previous generations of his family, of a tradition of service in the medical and teaching professions. He worked to repay that legacy in his time. His medical specialty was cardiology, but his loyalty and interest emphasized comprehensive and personal care for the sick."

The late W. B. Bean, in his obituary for Byrd S. Leavell, wrote, "A monument may mark the grave of those whom we ultimately forget, but the goodness and character of a fine physician remain without a carved stone, wrought in the stuff of other mens' lives and deeds." To perpetuate our veneration of the goodness and character of our beloved colleague and friend, the University of Virginia plans to establish an endowed chair, the Lockhart B. McGuire Professorship in Internal Medicine. Contributions to that fund are being sought from colleagues, patients, and friends; they should be earmarked for the Lockhart McGuire Endowed Professorship and mailed to the University of Virginia Medical Alumni Foundation, Box 324, School of Medicine, Charlottesville, Virginia 22908.

News of the death of a Medical Society of Virginia member may appear in these pages in either or both of two ways: 1) a brief notice written by staff giving factual information; 2) a memoir contributed by a physician who knew the deceased peer and writes a personal account of his or her life. If the memoir is contributed before the brief notice is inserted, it is published at once; if after, it is set in type to be published as space permits.

Memoir of C. H. Lippard 1923-1990

By C. H. Sackett, MD, Donald Shotton, MD,
G. E. Calvert, MD, and W. H. Barney, MD

Carroll Hoyt (Cullie) Lippard died in Lynchburg, Virginia, on February 13, 1990, at the age of 66. Dr. Lippard was born in Statesville, North Carolina, and was educated in the public schools of North Carolina, receiving his undergraduate degree at the University of North Carolina. He was awarded his MD degree from the University of Pennsylvania in 1947 and had his internship and residency training in obstetrics and



CARROLL H. LIPPARD, MD
1923-1990

gynecology at the University of Pennsylvania, his time there being interrupted by United States Naval Service in the Panama Canal Zone.

Dr. Lippard began his practice of obstetrics and gynecology in 1953 and continued until his retirement in 1988. He was a member and past president of the Lynchburg Academy of Medicine. He was also a member of the Medical Society of Virginia, the American Medical Association, the Southern Medical Association, the Virginia Ob-Gyn Society, the Southeastern Ob-Gyn Society, and the South Atlantic Association of Obstetricians and Gynecologists, serving as president of that group in 1978-79.

In 1975, Dr. Lippard was appointed by Gov. Mills E. Godwin to the State Board of Medical Examiners.

where he served with distinction until 1983, and was Board president from 1980-1982.

In addition to his medical interest, he had an outstanding concern for civic affairs. He was president of Friends of the Lynchburg Public Library, and his involvement led to the present library in 1967. In 1967-68, Dr. Lippard organized and was chairman of the Central Virginia Asian Affairs Committee, and for this work he received the Law Day Award from the Lynchburg Bar Association in 1968. Also in 1968, Dr. Lippard was elected to the Lynchburg City Council and served as vice-mayor of Lynchburg from 1970-72. He was the organizing chairman of the Lynchburg Bicentennial Commission.

Dr. Lippard's sports love was tennis, at which he excelled, and he was the first president of the Lynchburg Tennis Patrons Association. He was a life-long Lutheran and was active in Holy Trinity Lutheran Church, serving in many capacities, including president of the Church Council. He was also an accomplished musician, played the piano and sang in the Holy Trinity Church Choir for 35 years.

Dr. Lippard is survived by his wife, Sally Ford Lippard, and four children, Leslie Lippard Brown Hawes, Ann Lippard Drescher, Margaret Kimball Lippard, and Robert Carroll Lippard; and grandchildren, Megan, Molly Kate, and Matthew Brown; Cullen, John Brent and Christopher Corbin Drescher; and Brittany Gray and Robert Lippard.

Dr. Lippard was highly respected by his colleagues and his patients, and tributes following his death came from many walks of life. The Lynchburg newspaper editorialized that "from city library to City Hall to hospital delivery room, the memory of Dr. Carroll H. Lippard lingers as a kind man who believed in the innate goodness of people."

Colleagues reported that he brought a touch of class and gentility to everything he did, that he had an excellent mind, was a real scholar and a student of history and philosophy. One colleague described him as a "thoroughbred" and stated that "all people looked alike to him."

Cullie delivered more than 12,000 babies in his career and continued deliveries until the week before he retired.

Cullie's death is a loss to Lynchburg and to the medical profession, but both are better for his having passed this way.

Memoir of Ralph R. Landes 1911-1989

By B. R. Ashby, MD

Ralph R. Landes, MD, who founded the Danville Urologic Clinic and was the senior partner until his retirement in 1985, died October 5, 1989, in Chicago.

A native of Milwaukee, Wisconsin, Dr. Landes graduated from the University of Wisconsin and received his MD from Rush Medical College of the University of Chicago in 1939. He served his internship at Michael Reese Hospital and did his surgery and urology training there and at Boston City Hospital. During World War II he served as a medical officer with the 16th Evacuation Hospital in Africa and Italy and after discharge joined the medical staff of the Martinsville, West Virginia, Veterans Administration Hospital. In 1949 he moved to Danville to begin his private practice of urology.

Dr. Landes was president in 1960 of the medical staff at Danville's Memorial Hospital, and had served also as the hospital's chief of surgery (1965-1967) and urology (1960-1976). The library at the hospital was named the Ralph R. Landes Medical Library in his honor after his retirement. At the University of North Carolina, where he served as clinical professor of urology, his colleagues and former residents established the Ralph R. Landes Annual Lectureship. He also was a lecturer in medical history at the Bowman Gray School of Medicine of Wake Forest University. He was the author of more than 150 published articles and frequently presented papers at scientific meetings in this country and abroad.

He was president in 1968 of the Danville-Pittsylvania Academy of Medicine and in 1960 of the Virginia Urological Society. From 1965 to 1979 he served as historian of the American Urological Association. A fellow of the American College of Surgeons, he belonged also to the Medical Society of Virginia, American Medical Association, American Association of Clinical Urologists, American Academy of Pediatrics (urological fellow), the Society for Pediatric Urology, the Southern Medical Association, and the Société Internationale d'Urologie.

He was very active in community affairs and was on the board of trustees of Averett College in Danville, the Wayles Harrison Cancer Foundation, and the Science Museum of Virginia in Richmond. He was a member of the Church of the Epiphany in Danville.

He lived his retirement years in Danville, Chicago, and the mountains of Virginia in Monterey, where he was interred.

He is survived by his devoted wife, Elizabeth Lightner Landes, of Chicago and Monterey; a son, John L. Landes of Nashville, Tennessee; and, a daughter, Elizabeth M. Landes Levanstam, of Chicago.

Physician, scholar, historian, teacher, researcher,

colleague, humanitarian, husband, father, friend—Dr. Ralph R. Landes was the perfect example of "a man of all seasons," whose memory will long endure in the hearts of all who were privileged to know him.

Memoir of Harold Bassham 1936-1990

By J. Thomas Hulvey, MD

Dr. Harold Lee Bassham, age 54, died at Johnston Memorial Hospital in Abingdon, Virginia, on May 17, 1990, following a brief illness. He was the senior radiologist at Johnston Memorial, having entered private practice there in April 1971.

Dr. Bassham graduated from Centenary College in 1956. He received his degree in medicine from Louisiana State University School of Medicine in 1960. He interned at the U.S. Naval Hospital in Portsmouth, Virginia, from 1960-61, then entered the navy nuclear power and submarine medicine training program, which he completed in 1962. He served in the U.S. Naval Submarine Force, Atlantic, until 1964 and was a

member of the commissioning team of the nuclear submarine U.S.S. James Monroe. He received his residency training in radiology at the U.S. Naval Hospital, Oakland, California, the University of California at San Francisco, the Armed Forces Institute of Pathology, and the National Naval Medical Center, Bethesda, Maryland, completing his residency in 1967. He was a veteran of the Vietnam War, serving as chief of radiology aboard the U.S.S. Repose.

He was a diplomate of the American Board of Radiology and a fellow of the American College of Radiology and the Radiological Society of North America. He belonged to The Medical Society of Virginia, Southwestern Virginia Medical Society, and the Washington County Medical Society.

Lee was an exceptional radiologist and a cherished member of his hospital staff. He was a thoughtful, caring man who gave freely of his time and his many talents to those who sought his counsel. He was a devoted churchman, having served on the vestry of St. Thomas Episcopal Church, and he had worked with the Boy Scouts.

He is survived by his wife, Joyce; his sons, Randy, Steve, Mark and Phillip; his parents; and a sister. He will be remembered with a great sense of loss by his friends and colleagues.



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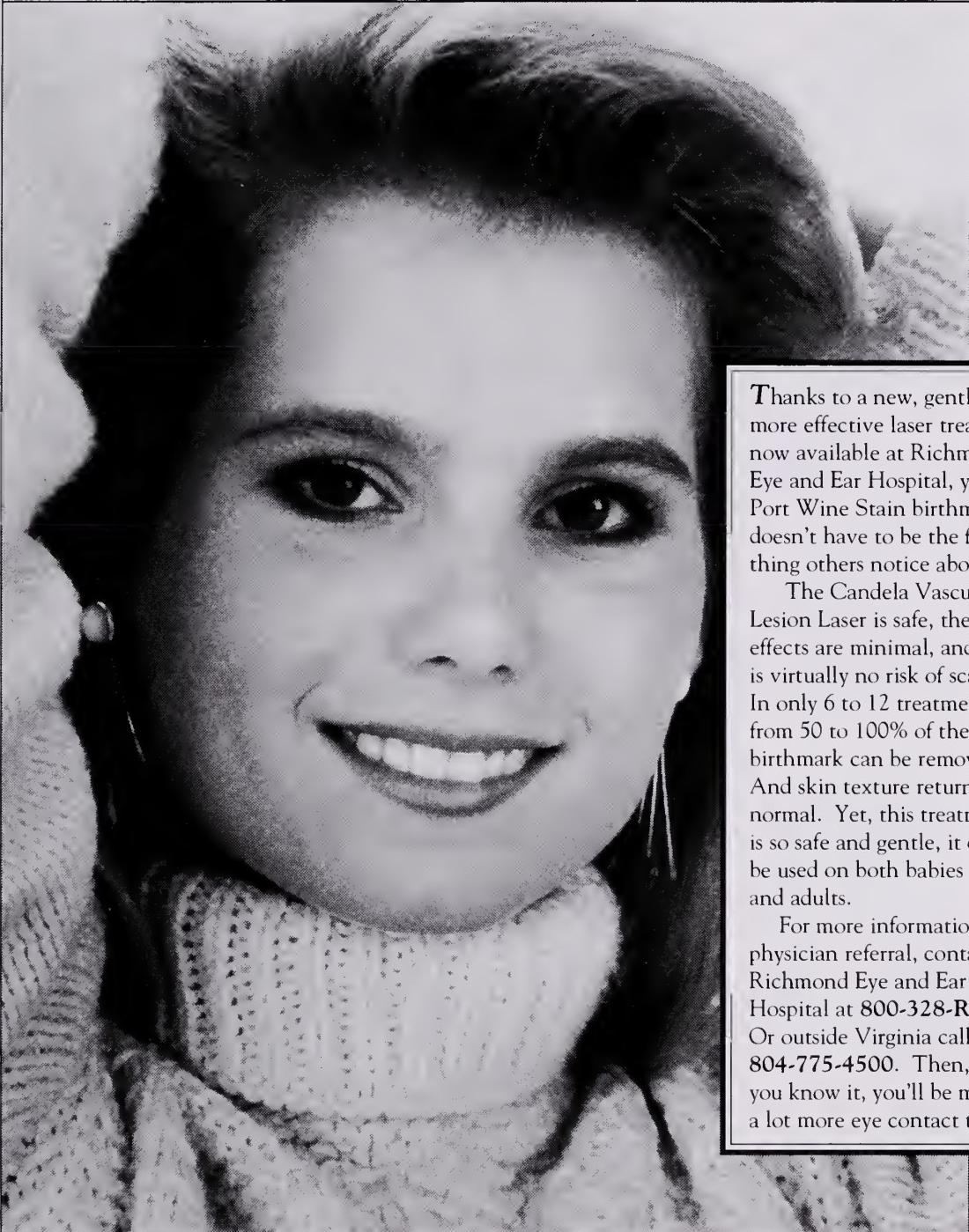
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Am Fam Phys 1987;36:133-140

Brief Summary.

Consult the package literature for prescribing information.
Indication: Lower respiratory infections, including pneumonia, caused by *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Streptococcus pyogenes* (group A β -hemolytic streptococci).

Contraindication: Known allergy to cephalosporins.

Warnings: CECLOL SHOULD BE ADMINISTERED CAUTIOUSLY TO PENICILLIN-SENSITIVE PATIENTS. PENICILLINS AND CEPHALOSPORINS SHOW PARTIAL CROSS-ALLERGENICITY. POSSIBLE REACTIONS INCLUDE ANAPHYLAXIS.

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Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics. It must be considered in differential diagnosis of antibiotic-associated diarrhea. Colon flora is altered by broad-spectrum antibiotic treatment, possibly resulting in antibiotic-associated colitis.

Precautions:

- Discontinue Ceclor in the event of allergic reactions to it.
- Prolonged use may result in overgrowth of non-susceptible organisms.
- Positive direct Coombs' tests have been reported during treatment with cephalosporins.
- Ceclor should be administered with caution in the presence of markedly impaired renal function. Although dosage adjustments in moderate to severe renal impairment are usually not required, careful clinical observation and laboratory studies should be made.
- Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.
- Safety and effectiveness have not been determined in pregnancy, lactation, and infants less than one month old. Ceclor penetrates mother's milk. Exercise caution in prescribing for these patients.

Adverse Reactions: (percentage of patients)

- Therapy-related adverse reactions are uncommon. Those reported include:
 - Hypersensitivity reactions have been reported in about 1.5% of patients and include morbilliform eruptions (1 in 100). Pruritus, urticaria, and positive Coombs' tests each occur in less than 1 in 200 patients. Cases of serum-sickness-like reactions have been reported with the use of Ceclor. These are characterized by findings of erythema multiforme, rashes, and other skin manifestations accompanied by arthritis/arthralgia, with or without fever, and differ from classic serum sickness in that there is infrequently associated lymphadenopathy and proteinuria, no circulating immune complexes, and no evidence to date of sequelae of the reaction. While further investigation is ongoing, serum-sickness-like reactions appear to be due to hypersensitivity and more often occur during or following a second (or subsequent) course of therapy with Ceclor. Such reactions have been reported more frequently in children than in adults with an overall occurrence ranging from 1 in 200 (0.5%) in one focused trial to 2 in 8,346 (0.024%) in overall clinical trials (with an incidence in children in clinical trials of 0.055%) to 1 in 38,000 (0.003%) in spontaneous event reports. Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy; occasionally these reactions have resulted in hospitalization, usually of short duration (median hospitalization = two to three days, based on postmarketing surveillance studies). In those requiring hospitalization, the symptoms have ranged from mild to severe at the time of admission with more of the severe reactions occurring in children. Antihistamines and glucocorticoids appear to enhance resolution of the signs and symptoms. No serious sequelae have been reported.
 - Stevens-Johnson syndrome, toxic epidermal necrolysis.

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and anaphylaxis have been reported rarely. Anaphylaxis may be more common in patients with a history of penicillin allergy.

- Gastrointestinal (mostly diarrhea): 2.5%
- Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment:
 - As with some penicillins and some other cephalosporins, transient hepatitis and cholestatic jaundice have been reported rarely.
 - Rarely, reversible hyperactivity, nervousness, insomnia, confusion, hypertension, dizziness, and somnolence have been reported.
 - Other: eosinophilia, 2%; genital pruritis or vaginitis, less than 1% and, rarely, thrombocytopenia and reversible interstitial nephritis.

Abnormalities in laboratory results of uncertain etiology

- Slight elevations in hepatic enzymes.
- Transient lymphocytosis, leukopenia, and, rarely, hemolytic anemia and reversible neutropenia.
- Rare reports of increased prothrombin time with or without clinical bleeding in patients receiving Ceclor and Coumadin concomitantly.
- Abnormal urinalysis; elevations in BUN or serum creatinine.
- Positive direct Coombs' test.
- False-positive tests for urinary glucose with Benedict's or Fehling's solution and Clinistest® tablets but not with Tes-Tape® (glucose enzymatic test strip, Lilly). PA 8791 AMP [021490 LRI]

Additional information available to the profession on request from Eli Lilly and Company, Indianapolis, Indiana 46285.



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BOOKS

Colonel Parke of Virginia: "The Greatest Hector in the Town," by Helen Hill Miller. Chapel Hill, North Carolina, Algonquin Books of Chapel Hill, 1990, 232 pages, illustrations, \$19.95 cloth.

For those of us in Virginia, it is always interesting to read of the major and minor characters forging the origins of our great country, starting with England's first permanent colony in America. Such a person was Daniel Parke, Jr., part-time roué, part-time adventurer, but always on top of things until he met his end in the Leeward Islands of the Caribbean at the hands of the islanders enraged at his adulterous ways.

This man managed to be the one to bring the news of the tremendous victory of John Churchill, Duke of Marlborough, and his combined forces from England, Holland and Germany against the combined armies of France and Bavaria. Parke delivered the news personally to Queen Anne and, for his labors, was given a diamond-encrusted gold locket. Parke put together, largely by inheritance, the single largest estate in York County, Virginia. This gave him entree into Virginia politics with an eventual seat on the Governor's Council, but never the governorship to which he aspired. His corrupt ways hampered him in Virginia as well as England, where he sought a seat in the House of Commons.

Failing to get the Royal Governor's appointment to Virginia, Parke turned to the Caribbean Islands; the future seemed bright there for England as the power of Spain declined. Here again Parke lost it all by his corrupt and immoral ways. He was Royal Governor of the Leeward Islands in the West Indies when he met his end.

Mrs. Miller is authoritative without being pedantic and focuses on data she has meticulously gathered into a decision tree and the issues are well addressed.



Daniel Parke, Jr.
From *Colonel Parke of Virginia*

The author describes a quidnunc of the times, with an eerie sense of déjà vu, as this rake goes from one adventure to another, seemingly caring not at all what he does to others to achieve his ends. His flawed character and eccentricities are clearly brought out by Mrs. Miller. The extensive research done by this remarkable writer, who was in her 91st year when this book was written, is a paradigm of critical social insights and analyses of seventeenth century doings which had such an impact on Virginia and its early development.

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MEETINGS

October 31

Risk Management and Medical Insurers (Medical Society of Virginia/Virginia Insurance Reciprocal/Medical Protective), the Homestead, Hot Springs. 4 credit hrs. Mrs. Evelyn Stockmar at MSV headquarters, 804-353-2721.

November 1-4

5th Annual Advances and Controversies in Internal Medicine Seminar (Lloyd Noland Hospital), the Greenbrier, White Sulphur Springs, West Virginia. Dr. George M. Converse, 205-783-5276 or -5121.

November 1-4

Principles and Practice of MRI (Johns Hopkins), Baltimore. 24 credit hrs. Fee: \$495. CME Office, 301-955-2959.

November 3

A Day on Foot, a Symposium on Foot Disorders (National Hospital for Orthopaedics/Rehabilitation), Arlington. Leslie Gilliam, 703-920-6700.

November 3-4

Hemodynamic Monitoring, Patient Care and Pulmonary Ar-

tery Catheterization (Johns Hopkins), Baltimore. 14 credit hrs. Fee: \$425. CME Office, 301-955-2959.

November 5

Advanced Pediatric Life Support Course (Johns Hopkins), Baltimore. 20 credit hrs. Fee: \$495. CME Office, 301-955-2959.

November 10

Managed Care: 1990s and Beyond (Eastern Virginia Medical School), Virginia Beach. 5 credit hrs. Fee: \$25. CME Office, 804-446-6140.

November 11-15

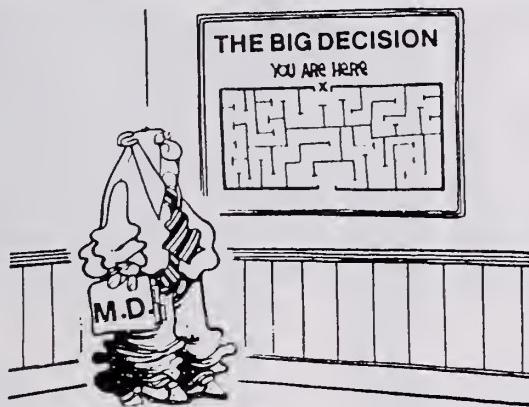
13th Edition, Practical Dermatology for the Primary Care Physician (Eastern Virginia Medical School), San Diego, California. 22 credit hrs. Fee: \$425. CME Office, 804-446-6140.

November 12-15

Primary Care Update (Interstate Postgraduate Medical Association), Walt Disney World Village, Lake Buena Vista, Florida. IPMA, 608-257-1401.

Continued on page 395.

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MORE MEETINGS

November 29-December 3

16th Annual Assembly Meeting, Hospital Medical Staff Section, American Medical Association, Orlando, Florida. 312-464-4754 or -4761.

December 7-8

7th Annual Virginia Impaired Health Professionals Conference (MSV Physicians' Health/Effectiveness Committee), Williamsburg. Jeanne Douglas, 804-353-2721.

January 24-26

25th Annual Meeting of the Neurosurgical Society of the Virginias, the Greenbrier, White Sulphur Springs, West Virginia. Dr. George Johnson, 804-320-2758.

January 27-February 1

Gastroenterology Update 1991 (Johns Hopkins/American College of Gastroenterology), Aspen/Snowmass, Colorado. 20 credit hrs. Fee: \$450. CME Office, 301-955-2959.

February 1-3

Annual Meeting of the Virginia Neurological Society, the Homestead, Hot Springs. Donna Scott, 804-353-2721.

February 28-March 3

Principles and Practice of Clinical MRI (Johns Hopkins),

Walt Disney World Village, Lake Buena Vista, Florida. 20 credit hrs. Fee: \$495. CME Office, 301-955-2959.

February 10-14

1991 Scientific Assembly of the Virginia Chapter, American College of Emergency Physicians, the Homestead, Hot Springs. 18 credit hrs. Gwen Messler Harry, 804-966-5966.

February 23-March 2

5th Annual Current Innovations in the Practice of Gastroenterology, (Georgetown University), Snowmass Village, Colorado. CME Office, 202-687-8735.

March 1-3

Virginia Pediatric Conference '91: Annual Meeting of the Virginia Chapter, American Academy of Pediatrics and the Virginia Pediatric Society, Boar's Head Inn, Charlottesville. 804-643-8130.

May 3-5

Annual Meeting of the Virginia Surgical Society, Williamsburg. Irving L. Kron, MD, 804-924-2158.

May 9-12

Annual Meeting of the Virginia Chapter, American College of Radiology, Williamsburg. Patricia R. Berry, 703-669-8312.



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Eric R. Powers, MD, Cardiology, Box 158-22, University of Virginia Health Sciences Center, Charlottesville VA 22908

Frederic B. Westervelt, MD, Nephrology, Box 133, University of Virginia Health Sciences Center, Charlottesville VA 22908

Alexandria Medical Society

James E. Burgess, MD, Neurosurgery, 719 South Lee Street, Alexandria VA 22314

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Wayne D. Old, MD, Cardiology, 1321 Smith Cove Circle, Virginia Beach VA 23455

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Phong Q. Nguyen, MD, Rheumatology, 1800 Town Center Parkway, Reston VA 22090

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Jeffrey B. Rich, MD, Cardiovascular Surgery, 400 West Brambleton Avenue, Norfolk VA 23510

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John W. Steffe, Jr., MD, Diagnostic Radiology, 2037 Crystal Spring Avenue SW, Roanoke VA 24014

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Karen A. Lyons, MD, Internal Medicine, 110 Houston Street, Lexington VA 24450

continued on page 398

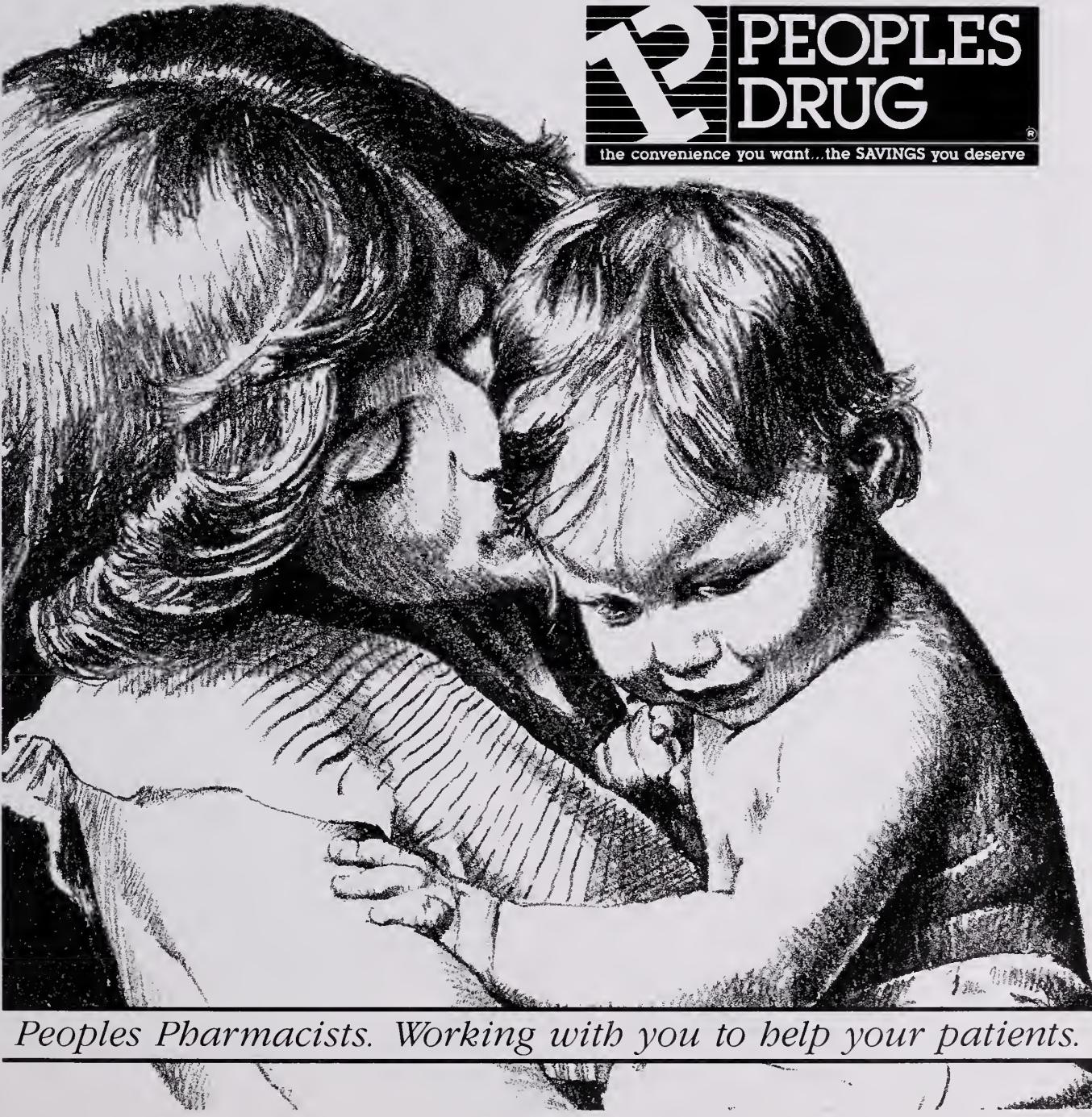
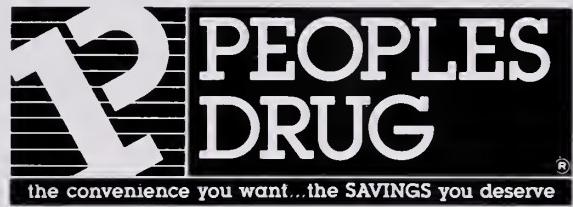
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Martha G. Sparrow, MD, Pediatrics, 10 Hickok Street, Christiansburg VA 24073

Donald Zedalis, MD, Allergy, 601 Colony Park, Blacksburg VA 24060

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Jan A. Janson, MD, Gastroenterology, 1101 First Colonial Road, Virginia Beach VA 23454

Meredith B. Rose, MD, Family Practice, 5320 Providence Road, Virginia Beach VA 23464

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Bruce M. Bradfield, MD, Family Practice, 2213 Stanley Avenue, Roanoke VA 24041

Karin W. Buettner, MD, Obstetrics/Gynecology, 9303 Newhall Road, Richmond VA 23229

John V. Conte, Jr., MD, General Surgery, 3103 S. Manchester Street, Falls Church VA 22044

Thomas J. Eichler, MD, Radiation Oncology, 1816 Oakdale Avenue, Richmond VA 23227

Thomas Florian, MD, Physical Medicine/Rehabilitation, 2524 Maplewood Avenue, Richmond VA 23228

Randolph A. Frank, Jr., MD, Psychiatry, Route 1, Box 26, Afton VA 22920

Phyllis R. Hope, MD, Pediatrics, 6145 Sylvan Street, Norfolk VA 23508

Joel C. Hutcheson, MD, General Surgery, PO Box 398, Blacksburg VA 24060

Lindsey A. Johnson, MD, Pediatrics, 2032 Denton Drive, Richmond VA 23235

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Brian C. McCormick, MD, Family Practice, 107 Moores Creek Drive, Tabb VA 23602

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CONTRAINDICATIONS:

L-thyroxine therapy is contraindicated in thyrotoxicosis, acute myocardial infarction and uncorrected adrenal insufficiency.

WARNINGS:

Drugs with thyroid hormone activity, alone or together with other therapeutic agents, have been used for the treatment of obesity. In euthyroid patients, doses within the range of daily hormonal requirements are ineffective for weight reduction. Larger doses may produce serious or even life-threatening manifestations of toxicity, particularly when given in association with sympathomimetic amines such as those used for cosmetic effects.

PRECAUTIONS:

Caution must be exercised in the administration of this drug to patients with cardiovascular disease. Development of chest pain or other aggravation of the cardiovascular disease requires a reduction of dosage.

Patients on thyroid preparations and parents of children on thyroid therapy should be informed that replacement therapy is to be taken essentially for life. They should immediately report during the course of therapy any signs or symptoms of thyroid hormone toxicity, e.g., chest pains, increased pulse rate, palpitations, excessive heat, intolerance, nervousness, or any other unusual event. In case of concomitant diabetes mellitus, the daily dosage of antidiabetic medication may need readjustment. In case of concomitant oral anticoagulant therapy, the prothrombin time should be measured frequently to determine if the dosage of oral anticoagulants is to be readjusted.

Partial loss of hair may be experienced by children in the first few months of thyroid therapy, but this is usually a transient phenomenon and later recovery is usually the rule.

Drug Interactions — In patients with diabetes mellitus, addition of thyroid hormone therapy may cause an increase in the required dosage of insulin or oral hypoglycemic agents.

Patients stabilized on oral anticoagulants who are found to require thyroid replacement therapy should be watched very closely when therapy is started.

Cholestyramine binds both T_4 and T_3 in the intestine, thus impairing absorption of these thyroid hormones. Four to five hours should elapse between administration of cholestyramine and thyroid hormones.

Estrogens tend to increase serum thyroxine-binding globulin (TBG). Patients without a functioning thyroid gland and who are on thyroid replacement therapy may need to increase their thyroid dose if estrogens or estrogen-containing oral contraceptives are given.

Drug/Laboratory Test Interactions — The following drugs or moieties are known to interfere with laboratory tests performed on patients taking thyroid hormone, androgens, corticosteroids, estrogens, oral contraceptives containing estrogens, iodine-containing preparations, and the numerous preparations containing salicylates.

Carcinogenesis, Mutagenesis, And Impairment of Fertility — A reported apparent association between prolonged thyroid therapy and breast cancer has not been confirmed. No confirmatory long-term studies in animals have been performed to evaluate carcinogenic potential, mutagenicity, or impairment of fertility in either males or females.

Pregnancy—Category A — The clinical experience to date does not indicate any adverse effect on fetuses when thyroid hormones are administered to pregnant women.

Nursing Mothers — Minimal amounts of thyroid hormones are excreted in human milk. Thyroid is not associated with serious adverse reactions and does not have a known tumorigenic potential. However, caution should be exercised when thyroid is administered to a nursing woman.

Pediatric Use — The incidence of congenital hypothyroidism is relatively high. Routine determinations of serum (T_4) and/or TSH is strongly advised in neonates in view of the deleterious effects of thyroid deficiency on growth and development.

ADVERSE REACTIONS:

Adverse reactions are due to overdosage and are those of induced hyperthyroidism.

OVERDOSEAGE — Excessive dosage of thyroid medication may result in symptoms of hyperthyroidism, which may not appear for one to three weeks after the dosage regimen is begun. The most common signs and symptoms of overdosage are weight loss, palpitation, nervousness, diarrhea or abdominal cramps, sweating, tachycardia, cardiac arrhythmias, angina pectoris, tremors, headache, insomnia, intolerance to heat and fever. If symptoms of overdosage appear, discontinue medication for several days and reinstitute treatment at a lower dosage level.

Complications as a result of the induced hypermetabolic state may include cardiac failure and death due to arrhythmia or failure.

Dosage should be reduced or therapy temporarily discontinued if signs and symptoms of overdosage appear.

Treatment of acute massive thyroid hormone overdose is aimed at reducing gastrointestinal absorption of the drugs and counteracting central and peripheral effects, mainly those of increased sympathetic activity. Measures to control fever, hypoglycemia, or fluid loss should be instituted if needed.

DOSAGE FORMS AVAILABLE:

LEVOXINE (L-thyroxine) tablets are supplied as oval, color coded, potency marked tablets in 10 strengths: 25 mcg (0.025 mg) — orange, 50 mcg (0.05 mg) — white, 75 mcg (0.075 mg) — purple, 100 mcg (0.1 mg) — yellow, 112 mcg (0.112 mg) — rose, 125 mcg (0.125 mg) — brown, 150 mcg (0.15 mg) — blue, 175 mcg (0.175 mg) — turquoise, 200 mcg (0.2 mg) — pink and 300 mcg (0.3 mg) — green, in bottles of 100 and 1000, and unit dose in cartons of 100 (10 strips of 10 tablets).

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WHO'S WHO

Dr. Percy Wootton of Virginia moved into the national spotlight at the American Medical Association's recent annual meeting in Chicago when he served as chairman of one of the meeting's three reference committees. Well schooled in the art of presiding, the Richmond cardiologist is a past president of the Medical Society of Virginia and current chairman of the Society's AMA delegation. For the AMA, he is a member of the Council on Legislation and has announced his candidacy, in the 1991 election, for the AMA Board of Trustees.

Dr. Raymond S. Brown has retired from his Gloucester Court House family practice. Plagued by severe arthritis and depleted by over 40 years of practicing solo, he feels the need, he says, to spend a lot of time in a rocking chair on the porch of his home in Zanoni. From Zanoni he can see the house where he was born. "I was born and raised and worked and will be buried within a 5-mile radius," he observes.

A graduate of the Medical College of Virginia, Dr. Brown spent three years in the Navy before starting up his practice in Gloucester. The minute his practice began, so, too, did his commitment to community service, and in 1977 the Medical Society of Virginia gave him its highest honor, the A. H. Robins Award for Community Service. Cited were his leadership roles in the Gloucester Rotary and Lions Clubs, the Ware Episcopal Church Men's Club, Virginia Tuberculosis Association, local chapter of the Red Cross, Gloucester Library Fund, and the F. N. Sanders Nursing Home. He has been president of the Mid-Tidewater Medical Society, the Hampton Roads Academy of Family Practice, and the Medical Society of Virginia, and is currently a member of the

Virginia delegation to the AMA and this journal's Editorial Board.

After a two-year search, Dr. Brown found a successor in Chicago. He is Dr. Mark Shawnik, who moved his wife and three children to Virginia's Northern Neck and took over the practice early in August.

New president of the Seaboard Medical Association is **Dr. Roger T. Gregory**, Norfolk cardiovascular surgeon. Other Virginians elected to office with him are **Dr. Alvin J. Ciccone**, Norfolk, second vice president, and **Dr. Ronald K. Davis**, Richmond, fourth vice president.

Dr. Frank S. Blanton, Jr., Bristol, was elected president of the Virginia Chapter, American College of Surgeons, during the chapter's annual meeting in Norfolk.

A Richmond surgeon, **Dr. Wyatt S. Beazley III**, heads the Virginia Surgical Society as president for 1990-91. His election occurred at the society's annual meeting in Hot Springs.



Dr. Wootton

Virginia anesthesiologists have elected as their new president **Dr. William P. Arnold III**, of the University of Virginia School of Medicine, Charlottesville.

When Eastern Virginia Medical School's Faculty Achievement Awards for 1990 were handed out, two of the five winners were physicians. They are **Dr. David B. Propert**, professor of internal medicine, whose award was for achievement in teaching in the clinical sciences, and **Dr. Clai-borne W. Fitchett**, professor of surgery, whose award honored achievement by community faculty.

The names of two Medical Society of Virginia members showed up among Gov. L. Douglas Wilder's appointments: **Dr. David W. Branch**, Roanoke, was appointed a member of the State Board of Medicine, and **Dr. C. M. Kinloch Nelson**, Richmond, was reappointed a member of the board of directors of the Virginia Birth-Related Neurological Injury Compensation Program.

Across the Bay from Gloucester, on Virginia's Eastern Shore, **Dr. Walter A. Eskridge** couldn't give his practice away when he retired.

"A few people came to look it over," he related, "and one even kept coming back over a period of



Dr. Brown

three or four weeks, but he finally decided against it." So Dr. Eskridge closed his office in Parksley, sold the equipment, and set about sorting/storing a four-decade accumulation of medical records. Parksley had no doctor when he set up practice there in 1952 and now has none again.

Born and reared in West Virginia, Dr. Eskridge took his MD degree and also interned at the Medical College of Virginia. His training complete, he looked for a place "where they really needed a doctor," and found it in Parksley. By the time he retired Dr. Parker C. Dooley, Eastern Shore internist, commented to reporter Nancy Namoski of the *Eastern Shore News* that "we would need three or four doctors to replace Dr. Eskridge. I don't know if anyone is willing to work that hard."

In addition to his clinical case load, Dr. Eskridge served as mayor of Parksley and was for many years the Accomack County medical examiner and the physician for the Accomack County Nursing Home. He was a member of the Virginia State Board of Medicine 1973-78, has held all available offices, including the presidency, of the Accomack County Medical Society, and this year for the umpteenth time will be a delegate to the Medical Society of Virginia's annual meeting.

The Accomack County Board of Supervisors honored his retirement by giving him a plaque of appreciation and a standing ovation, and the townsfolk turned out for "Walter Eskridge Day in the Park."

Dr. Charles H. Robertson, Jr., Richmond pulmonary specialist is the new president of the American Lung Association of Virginia.

In ceremonies at the AMA National Congress on Adolescent Health in Chicago, **Dr. Richard H. Schwartz** of Vienna, Virginia, received an award for distinguished service on behalf of America's youth. Dr. Schwartz "combines the private practice of pediatrics with a dedication to teaching and the prevention of drug and alcohol abuse."



Dr. Eskridge

said the Adolescent Health award citation, and "has distinguished himself in service to youth." A member of the American Academy of Pediatric's Task Force on Substance Abuse, Dr. Schwartz was the 1989 recipient of the Academy's Practitioner Research Award.

As president of the Historical Society of Mecklenburg County, **Dr. William A. Shelton** is heading up the restoration of "Elm Hill," an 18th century estate occupying an important role in the Southside county's history. Built in the mid-1700s by a Prince George County merchant, Elm Hill became the home of Sir Peyton Skipwith in the 1760s and stayed in the Skipwith family until 1865. The Association for the Preservation of Virginia Antiquities came into possession of it in 1982 and turned the deed over to the Mecklenburg society in 1988.

Three Medical Society of Virginia members were among the 118 physicians inducted as fellows of the American College of Critical Care Medicine at its meeting in San Francisco. They are **Dr. L. Delano Britt**, Norfolk; **Dr. Russell P. Seneca**, Annandale; and **Dr. John D. Ward**, Richmond.

Under construction in Norfolk is a new \$12.5 million facility to house the institute founded by **Drs. How-**

ard and Georgeanna Jones, whose work in reproductive medicine at Eastern Virginia Medical School is known 'round the world. Currently, clinical and research operations for the Jones Institute are located on separate campuses; the new 60,000-sq-ft, 4-story building will join them under one roof.

Dr. Morton P. Chiles, Culpeper, was named 1990 Physician of the Year at Culpeper Memorial Hospital for "special contributions to the community and to health care," said the hospital president, Bill Gravely, in conferring the award. "He has balanced his volunteer responsibilities with his medical practice in an exemplary way." Dr. Chiles is a member of the Culpeper County School Board and participates in a youth group at his church.

The Virginia Academy of Family Practice has named **Dr. James B. Harris** of Blackstone its Family Physician of the Year. The honor was bestowed at the Academy's annual meeting in Virginia Beach. A practicing family physician since 1960, Dr. Harris is well-known to Virginia's medical community for his leadership role in the Blackstone Family Practice Residency Program, through which 100 young doctors have been trained. He is a big man in the Blackstone community, too—the town's mayor since 1982 and president of the Blackstone Rotary Club.

The nation's veterans' hospitals have a new medical director. He is **Dr. James W. Holsinger, Jr.**, cardiologist, who was director of the McGuire Veterans Administration Medical Center in Richmond from 1981 until he departed to Washington. **Dr. John T. Farrar**, chief of staff at the Richmond VA Hospital for 11 years, was appointed Dr. Holsinger's associate deputy.

Photo of Dr. Wootton, Joe Fletcher for the AMA; Dr. Brown, courtesy the *Newport News Daily Press*; Dr. Eskridge, courtesy the *Eastern Shore News*.

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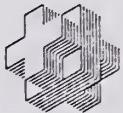
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References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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Contraindications: VASOTEC® (Enalapril Maleate, MSD) is contraindicated in patients who are hypersensitive to this product and in patients with a history of angioedema related to previous treatment with an ACE inhibitor.

Warnings: *Angioedema.* Angioedema of the face, extremities, lips, tongue, glottis, and/or larynx has been reported in patients treated with ACE inhibitors, including VASOTEC. In such cases, VASOTEC should be promptly discontinued and the patient carefully observed until the swelling disappears. In instances where swelling has been confined to the face and lips, the condition has generally resolved without treatment, although antihistamines have been useful in relieving symptoms. Angioedema associated with laryngeal edema may be fatal. **Where there is involvement of the tongue, glottis, or larynx likely to cause airway obstruction, appropriate therapy, e.g., subcutaneous epinephrine solution 1:1000 (0.3 mL to 0.5 mL), should be promptly administered.** (See ADVERSE REACTIONS.)

Hypotension: Excessive hypotension is rare in uncomplicated hypertensive patients treated with VASOTEC alone. Patients with heart failure given VASOTEC commonly have some reduction in blood pressure, especially with the first dose, but discontinuation of therapy for continuing symptomatic hypotension usually is not necessary when dosing instructions are followed; caution should be observed when initiating therapy. (See DOSAGE AND ADMINISTRATION.) Patients at risk for excessive hypotension, sometimes associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death, include those with the following conditions or characteristics: heart failure, hypotension, high-dose diuretic therapy, recent intensive diuresis or increase in diuretic dose, renal dialysis, or severe volume and/or salt depletion of any etiology. It may be advisable to eliminate the diuretic (except in patients with heart failure), reduce the diuretic dose, or increase salt intake cautiously before initiating therapy with VASOTEC in patients at risk for excessive hypotension who are able to tolerate such adjustments. (See PRECAUTIONS, Drug Interactions and ADVERSE REACTIONS.) In patients at risk for excessive hypotension, therapy should be started under very close medical supervision and such patients should be followed closely for the first two weeks of treatment and whenever the dose of enalapril and/or diuretic is increased. Similar considerations may apply to patients with ischemic heart disease or cardiovascular disease in whom an excessive fall in blood pressure could result in a myocardial infarction or cerebrovascular accident. If excessive hypotension occurs, the patient should be placed in the supine position and, if necessary, receive an intravenous infusion of normal saline. A transient hypotensive response is not a contraindication to further doses of VASOTEC, which usually can be given without difficulty once the blood pressure has stabilized. If symptomatic hypotension develops, a dose reduction or discontinuation of VASOTEC or concomitant diuretic may be necessary.

Neutropenia/Agranulocytosis: Another ACE inhibitor, captopril, has been shown to cause agranulocytosis and bone marrow depression, rarely in uncomplicated patients but more frequently in patients with renal impairment, especially if they also have a collagen vascular disease. Available data from clinical trials of enalapril are insufficient to show that enalapril does not cause agranulocytosis at similar rates. Foreign marketing experience has revealed several cases of neutropenia or agranulocytosis in which a causal relationship to enalapril cannot be excluded. Periodic monitoring of white blood cell counts in patients with collagen vascular disease and renal disease should be considered.

Precautions: General. Impaired Renal Function: As a consequence of inhibiting the renin-angiotensin-aldosterone system, changes in renal function may be anticipated in susceptible individuals. In patients with severe heart failure whose renal function may depend on the activity of the renin-angiotensin-aldosterone system, treatment with ACE inhibitors, including VASOTEC, may be associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death.

In clinical studies in hypertensive patients with unilateral or bilateral renal artery stenosis, increases in blood urea nitrogen and serum creatinine were observed in 20% of patients. These increases were almost always reversible upon discontinuation of enalapril and/or diuretic therapy. In such patients, renal function should be monitored during the first few weeks of therapy.

Some patients with hypertension or heart failure with no apparent preexisting renal vascular disease have developed increases in blood urea and serum creatinine, usually minor and transient, especially when VASOTEC has been given concomitantly with a diuretic. This is more likely to occur in patients with preexisting renal impairment. Dosage reduction and/or discontinuation of the diuretic and/or VASOTEC may be required.

Evaluation of patients with hypertension or heart failure should always include assessment of renal function. (See DOSAGE AND ADMINISTRATION.)

Hyperkalemia: Elevated serum potassium (>5.7 mEq/L) was observed in approximately 1% of hypertensive patients in clinical trials. In most cases these were isolated values which resolved despite continued therapy. Hyperkalemia was a cause of discontinuation of therapy in 0.28% of hypertensive patients. In clinical trials in heart failure, hyperkalemia was observed in 3.8% of patients, but was not a cause for discontinuation.

Risk factors for the development of hyperkalemia include renal insufficiency, diabetes mellitus, and the concomitant use of potassium-sparing diuretics, potassium supplements, and/or potassium-containing salt substitutes, which should be used cautiously, if at all, with VASOTEC. (See Drug Interactions.)

Surgery/Anesthesia: In patients undergoing major surgery or during anesthesia with agents that produce hypotension, enalapril may block angiotensin II formation secondary to compensatory renin release. If hypotension occurs and is considered to be due to this mechanism, it can be corrected by volume expansion.

Information for Patients:

Angioedema: Including laryngeal edema, may occur especially following the first dose of enalapril. Patients should be so advised and told to report immediately any signs or symptoms suggesting angioedema (swelling of face, extremities, eyes, lips, tongue, difficulty in swallowing or breathing) and to take no more drug until they have consulted with the prescribing physician.

Hypotension: Patients should be cautioned to report lightheadedness, especially during the first few days of therapy. If actual syncope occurs, the patients should be told to discontinue the drug until they have consulted with the prescribing physician.

All patients should be cautioned that excessive perspiration and dehydration may lead to an excessive fall in blood pressure because of reduction in fluid volume. Other causes of volume depletion such as vomiting or diarrhea may also lead to a fall in blood pressure; patients should be advised to consult with the physician.

Hyperkalemia: Patients should be told not to use salt substitutes containing potassium without consulting their physician.

Neutropenia: Patients should be told to report promptly any indication of infection (e.g., sore throat, fever) which may be a sign of neutropenia.

NOTE: As with many other drugs, certain advice to patients being treated with enalapril is warranted. This information is intended to aid in the safe and effective use of this medication. It is not a disclosure of all possible adverse or intended effects.

Drug Interactions:

Hypotension: Patients on Diuretic Therapy: Patients on diuretics and especially those in whom diuretic therapy was recently instituted may occasionally experience an excessive reduction of blood pressure after initiation of therapy with enalapril. The possibility of hypotensive effects with enalapril can be minimized by either discontinuing the diuretic or increasing the salt intake prior to initiation of treatment with enalapril. If it is necessary to continue the diuretic, provide close medical supervision after the initial dose for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and DOSAGE AND ADMINISTRATION.)

Agents Causing Renin Release: The antihypertensive effect of VASOTEC is augmented by antihypertensive agents that cause renin release (e.g., diuretics).

Other Cardiovascular Agents: VASOTEC has been used concomitantly with beta-adrenergic-blocking agents, methyldopa, nitrates, calcium-blocking agents, hydralazine, prazosin, and digoxin without evidence of clinically significant adverse interactions.

Agents Increasing Serum Potassium: VASOTEC attenuates potassium loss caused by thiazide-type diuretics. Potassium-sparing diuretics (e.g., spironolactone, triamterene, or amiloride), potassium supplements, or potassium-containing salt substitutes may lead to significant increases in serum potassium. Therefore, if concomitant use of these agents is indicated because of demonstrated hypokalemia, they should be used with caution and with frequent monitoring of serum potassium. Potassium-sparing agents should generally not be used in patients with heart failure receiving VASOTEC.

Lithium: Lithium toxicity has been reported in patients receiving lithium concomitantly with drugs which cause elimination of sodium, including ACE inhibitors. A few cases of lithium toxicity have been reported in patients receiving concomitant VASOTEC and lithium and were reversible upon discontinuation of both drugs. It is recommended that serum lithium levels be monitored frequently if enalapril is administered concomitantly with lithium.

Pregnancy - Category C: There was no fetotoxicity or teratogenicity in rats treated with up to 200 mg/kg/day of enalapril (333 times the maximum human dose). Fetotoxicity, expressed as a decrease in average fetal weight, occurred in rats given 1200 mg/kg/day of enalapril but did not occur when these animals were supplemented with saline. Enalapril was not teratogenic in rabbits. However, maternal and fetal toxicity occurred in some rabbits at doses of 1 mg/kg/day or more. Saline supplementation prevented the maternal and fetal toxicity seen at doses of 3 and 10 mg/kg/day, but not at 30 mg/kg/day (50 times the maximum human dose).

Radioactivity was found to cross the placenta following administration of labeled enalapril to pregnant hamsters.

There are no adequate and well-controlled studies of enalapril in pregnant women. However, data are available that show enalapril crosses the human placenta. Because the risk of fetal toxicity with the use of ACE inhibitors has not

been clearly defined, VASOTEC® (Enalapril Maleate, MSD) should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Postmarketing experience with all ACE inhibitors thus far suggests the following with regard to pregnancy outcome. inadvertent exposure limited to the first trimester of pregnancy has not been reported to affect fetal outcome adversely. Fetal exposure during the second and third trimesters of pregnancy has been associated with fetal and neonatal morbidity and mortality.

When ACE inhibitors are used during the later stages of pregnancy, there have been reports of hypotension and decreased renal perfusion in the newborn. Oligohydramnios in the mother has also been reported, presumably representing decreased renal function in the fetus. Infants exposed *in utero* to ACE inhibitors should be closely observed for hypotension, oliguria, and hyperkalemia. If oliguria occurs, attention should be directed toward support of blood pressure and renal perfusion with the administration of fluids and pressors as appropriate. Problems associated with prematurity such as patent ductus arteriosus have occurred in association with maternal use of ACE inhibitors, but it is not clear whether they are related to ACE inhibition, maternal hypertension, or the underlying prematurity.

Nursing Mothers: Milk in lactating rats contains radioactivity following administration of ¹⁴C enalapril maleate. It is not known whether this drug is secreted in human milk. Because many drugs are secreted in human milk, caution should be exercised when VASOTEC is given to a nursing mother.

Pediatric Use: Safety and effectiveness in children have not been established.

Adverse Reactions: VASOTEC has been evaluated for safety in more than 10,000 patients, including over 1000 patients treated for one year or more. VASOTEC has been found to be generally well tolerated in controlled clinical trials involving 2987 patients.

HYPERTENSION: The most frequent clinical adverse experiences in controlled trials were headache (5.2%), dizziness (4.3%), and fatigue (3%).

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in controlled clinical trials were: diarrhea (1.4%), nausea (1.4%), rash (1.4%), cough (1.3%), orthostatic effects (1.2%), and asthenia (1.1%).

HEART FAILURE: The most frequent clinical adverse experiences in both controlled and uncontrolled trials were: dizziness (7.9%), hypotension (6.7%), orthostatic effects (2.2%), syncope (2.2%), cough (2.2%), chest pain (2.1%), and diarrhea (2.1%).

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in both controlled and uncontrolled clinical trials were: fatigue (1.8%), headache (1.8%), abdominal pain (1.6%), asthenia (1.6%), orthostatic hypotension (1.6%), vertigo (1.6%), angina pectoris (1.5%), nausea (1.3%), vomiting (1.3%), bronchitis (1.3%), dyspepsia (1.3%), urinary tract infection (1.3%), rash (1.3%), and myocardial infarction (1.2%).

Other serious clinical adverse experiences occurring since the drug was marketed or adverse experiences occurring in 0.5% to 1% of patients with hypertension or heart failure in clinical trials in order of decreasing severity within each category

Cardiovascular: Cardiac arrest, myocardial infarction or cerebrovascular accident, possibly secondary to excessive hypotension in high-risk patients (see WARNINGS, Hypotension); pulmonary embolism and infarction; pulmonary edema; rhythm disturbances; atrial fibrillation; palpitation.

Digestive: Ileus, pancreatitis, hepatitis (hepatocellular or cholestatic jaundice), melena, anorexia, dyspepsia, constipation, glossitis, stomatitis, dry mouth.

Musculoskeletal/ Muscle cramps.

Nervous/Psychiatric: Depression, confusion, ataxia, somnolence, insomnia, nervousness, paresthesia.

Urogenital: Renal failure, oliguria, renal dysfunction (see PRECAUTIONS and OOSAGE AND ADMINISTRATION).

Respiratory: Bronchospasm, rhinorrhea, sore throat and hoarseness, asthma, upper respiratory infection.

Skin: Exfoliative dermatitis, toxic epidermal necrolysis, Stevens-Johnson syndrome, herpes zoster, erythema multiforme, urticaria, pruritis, alopecia, flushing, hyperhidrosis.

Special Senses: Blurred vision, taste alteration, anosmia, tinnitus, conjunctivitis, dry eyes, tearing.

A symptom complex has been reported which may include a positive ANA, an elevated erythrocyte sedimentation rate, arthralgias/arthritis, myalgias, fever, serositis, vasculitis, leukocytosis, eosinophilia, photosensitivity, rash, and other dermatologic manifestations.

Angioedema: Angioedema has been reported in patients receiving VASOTEC (0.2%). Angioedema associated with laryngeal edema may be fatal. If angioedema of the face, extremities, lips, tongue, glottis, and/or larynx occurs, treatment with VASOTEC should be discontinued and appropriate therapy instituted immediately. (See WARNINGS.)

Hypotension: In the hypertensive patients, hypotension occurred in 0.9% and syncope occurred in 0.5% of patients following the initial dose or during extended therapy. Hypotension or syncope was a cause for discontinuation of therapy in 0.1% of hypertensive patients. In heart failure patients, hypotension occurred in 6.7% and syncope occurred in 2.2% of patients. Hypotension or syncope was a cause for discontinuation of therapy in 1.9% of patients with heart failure. (See WARNINGS.)

Clinical Laboratory Test Findings:

Serum Electrolytes: Hyperkalemia (see PRECAUTIONS), hyponatremia.

Creatinine, Blood Urea Nitrogen: In controlled clinical trials, minor increases in blood urea nitrogen and serum creatinine, reversible upon discontinuation of therapy, were observed in about 0.2% of patients with essential hypertension treated with VASOTEC alone. Increases are more likely to occur in patients receiving concomitant diuretics or in patients with renal artery stenosis (see PRECAUTIONS). In patients with heart failure who were also receiving diuretics with or without digitalis, increases in blood urea nitrogen or serum creatinine, usually reversible upon discontinuation of VASOTEC and/or other concomitant diuretic therapy, were observed in about 11% of patients. Increases in blood urea nitrogen or creatinine were a cause for discontinuation in 1.2% of patients.

Hemoglobin and Hematocrit: Small decreases in hemoglobin and hematocrit (mean decreases of approximately 0.3-1% and 1.0 vol %, respectively) occur frequently in either hypertensive or heart failure patients treated with VASOTEC but are rarely of clinical importance unless another cause of anemia is present. In clinical trials, less than 0.1% of patients discontinued therapy due to anemia.

Other (Causal Relationship Unknown): In marketing experience, rare cases of neutropenia, thrombocytopenia, and bone marrow depression have been reported. A few cases of hemolytic anemia have been reported in patients with G6PD deficiency.

Liver Function Tests: Elevations of liver enzymes and/or serum bilirubin have occurred.

Dosage and Administration: **Hypertension:** In patients who are currently being treated with a diuretic, symptomatic hypotension occasionally may occur following the initial dose of VASOTEC. The diuretic should, if possible, be discontinued for two to three days before beginning therapy with VASOTEC to reduce the likelihood of hypotension. (See WARNINGS.) If the patient's blood pressure is not controlled with VASOTEC alone, diuretic therapy may be resumed. If the diuretic cannot be discontinued, an initial dose of 2.5 mg should be used under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and PRECAUTIONS, Drug Interactions.)

The recommended initial dose in patients not on diuretics is 5 mg once a day. Dosage should be adjusted according to blood pressure response. The usual dosage range is 10 to 40 mg per day administered in a single dose or in two divided doses. In some patients treated once daily, the antihypertensive effect may diminish toward the end of the dosing interval. In such patients, an increase in dosage or twice-daily administration should be considered. If blood pressure is not controlled with VASOTEC alone, a diuretic may be added.

Concomitant administration of VASOTEC with potassium supplements, potassium salt substitutes, or potassium-sparing diuretics may lead to increases of serum potassium (see PRECAUTIONS).

Dosage Adjustment in Hypertensive Patients with Renal Impairment: The usual dose of enalapril is recommended for patients with a creatinine clearance > 30 mL/min (serum creatinine of up to approximately 3 mg/dL). For patients with creatinine clearance ≤ 30 mL/min (serum creatinine ≥ 3 mg/dL), the first dose is 2.5 mg once daily. The dosage may be titrated upward until blood pressure is controlled or to a maximum of 40 mg daily.

Hypertension: VASOTEC is indicated as adjunctive therapy with diuretics and digitalis. The recommended starting dose is 2.5 mg once or twice daily. After the initial dose of VASOTEC, the patient should be observed under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and PRECAUTIONS, Drug Interactions.) Possibly, the dose of the diuretic should be reduced, which may diminish the likelihood of hypotension. The appearance of hypotension after the initial dose of VASOTEC does not preclude subsequent careful dose titration with the drug, following effective management of the hypotension. The usual therapeutic dosing range for the treatment of heart failure is 5 to 20 mg daily given in two divided doses. The maximum daily dose is 40 mg. Once-daily dosing has been effective in a controlled study, but nearly all patients in this study were given 40 mg, the maximum recommended daily dose, and there has been much more experience with twice-daily dosing. In addition, in a placebo-controlled study which demonstrated reduced mortality in patients with severe heart failure (NYHA Class IV), patients were treated with 2.5 to 40 mg per day of VASOTEC, almost always administered in two divided doses. (See CLINICAL PHARMACOLOGY, Pharmacodynamics and Clinical Effects.) Dosage may be adjusted depending upon clinical or hemodynamic response. (See WARNINGS.)

Dosage Adjustment in Patients with Heart Failure and Renal Impairment or Hyponatremia: In patients with heart failure who have hyponatremia (serum sodium < 130 mEq/L) or with serum creatinine > 1.6 mg/dL, therapy should be initiated at 2.5 mg daily under close medical supervision. (See DOSAGE AND ADMINISTRATION, Heart Failure, WARNINGS, and PRECAUTIONS, Drug Interactions.) The dose may be increased to 2.5 mg b.i.d., then 5 mg b.i.d. and higher as needed, usually at intervals of four days or more, if at the time of dosage adjustment there is not excessive hypotension or significant deterioration of renal function. The maximum daily dose is 40 mg.

For more detailed information, consult your MSD Representative or see Prescribing Information, Merck Sharp & Dohme Division of Merck & Co., Inc., West Point, PA 19486.

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THERAPY THAT MAY BE
AS SILENT AS
HYPERTENSION ITSELF

VASOTEC is generally well tolerated and not characterized by certain undesirable effects associated with selected agents in other antihypertensive classes.

VASOTEC is contraindicated in patients who are hypersensitive to this product and in patients with a history of angioedema related to previous treatment with an ACE inhibitor. A diminished antihypertensive effect toward the end of the dosing interval can occur in some patients.

For a Brief Summary of Prescribing Information, please see the last page of this advertisement.



FOR MANY
HYPERTENSIVE PATIENTS
ONCE-A-DAY

VASOTEC®
(ENALAPRIL MALEATE | MSD)

VIRGINIA MEDICAL

A PUBLICATION OF THE MEDICAL SOCIETY OF VIRGINIA

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b—book review	*—medical article
ed—editorial	mb—medibytes
h—history	pv—point of view
in—interview	s—speech
le—letter to editor	sa—special article
m—memoir	sr—special report
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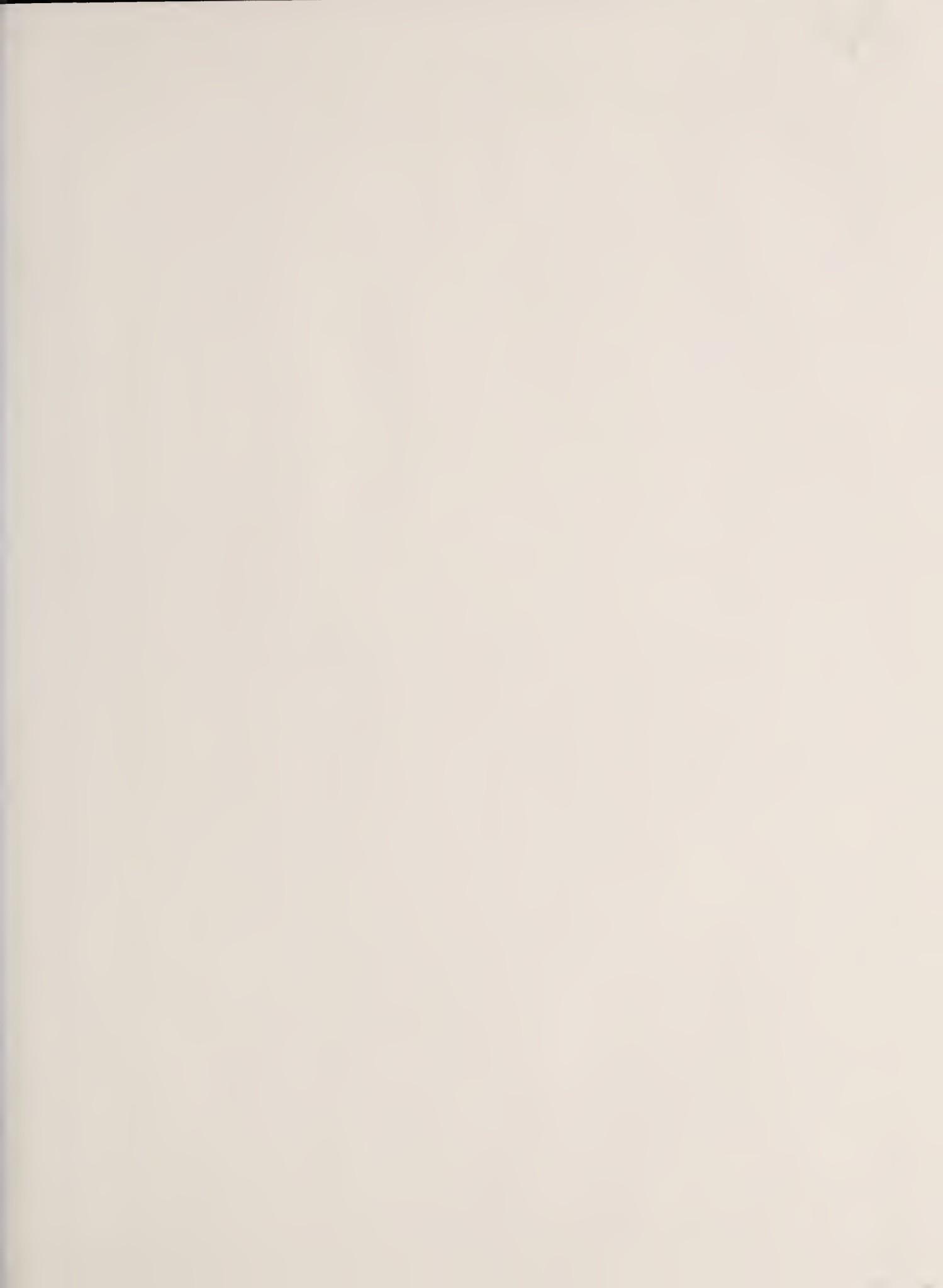
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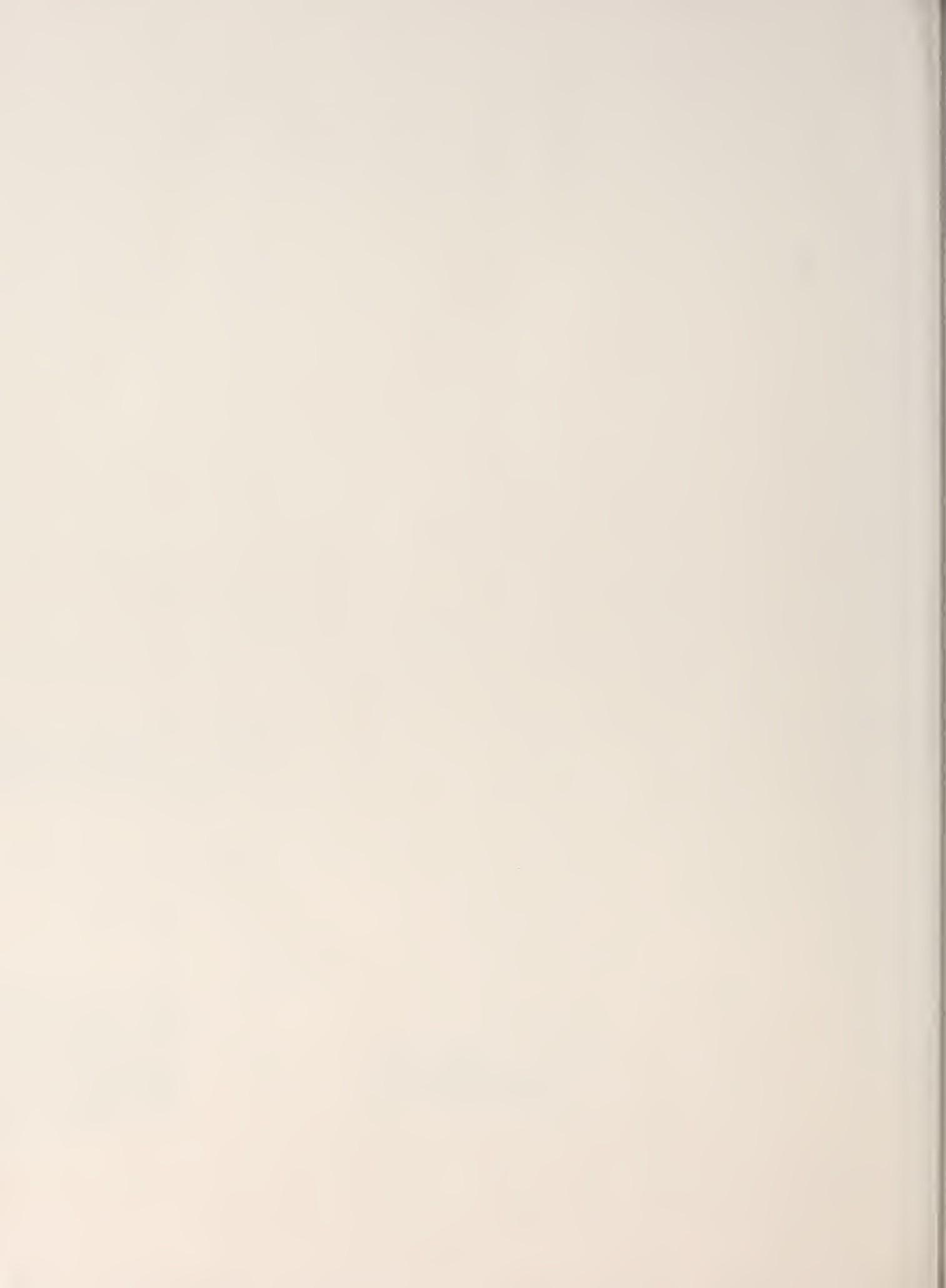


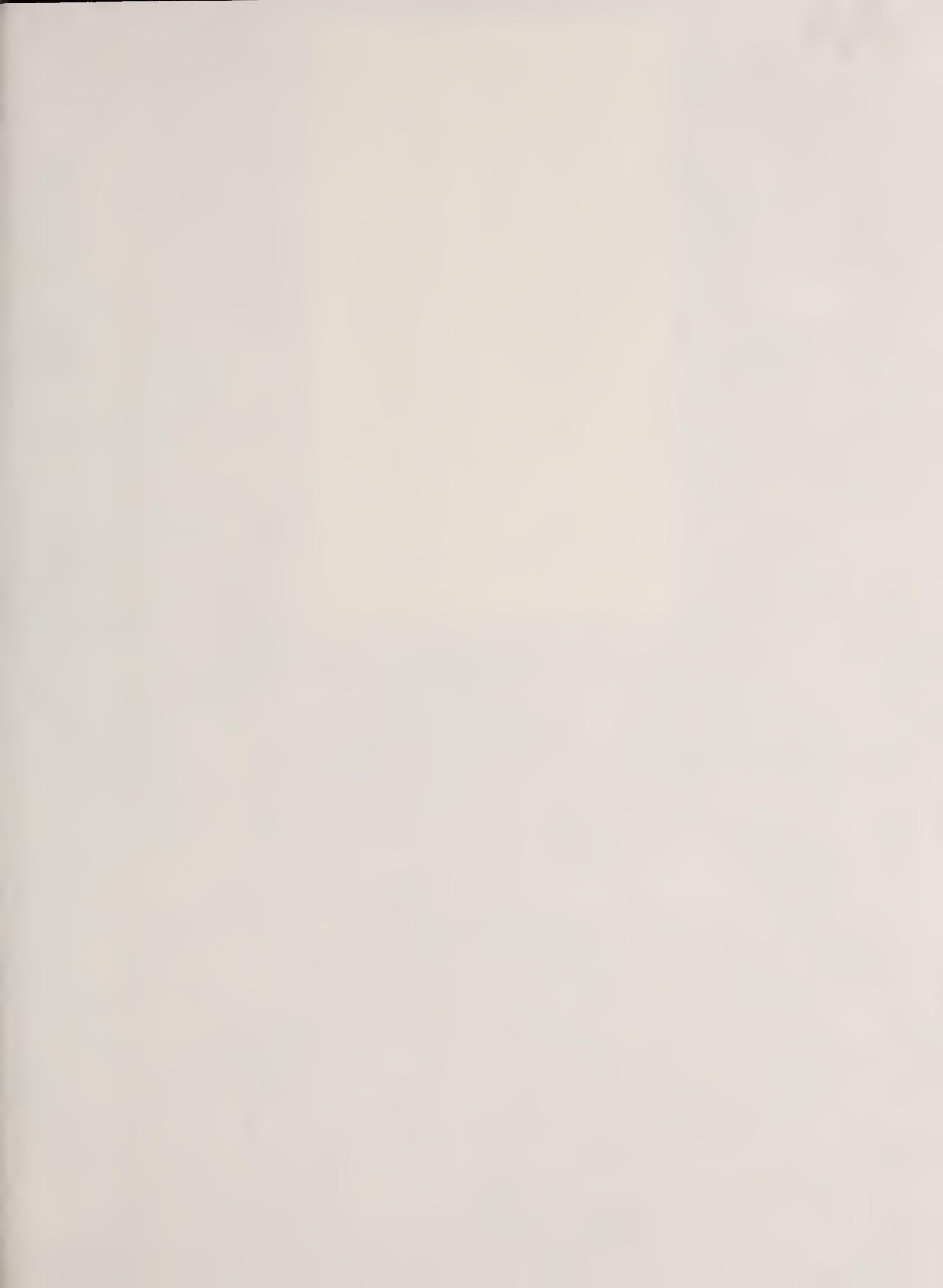
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